INCIDENCE AND OUTCOME OF

ILEAL PERFORATION

AMONG HOLLOW VISCUS PERFORATION

Dissertation submitted

To

THE TAMILNADU Dr. M. G. R. MEDICAL UNIVERSITY,
CHENNAI

With partial fulfilment of the regulations
For the award of the degree of

BRANCH - I M.S (GENERAL SURGERY)

APRIL 2014

GOVERNMENT KILPAUK MEDICAL COLLEGE & HOSPITAL

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Dr. SARAVANAN. M

On

"INCIDENCE AND OUTCOME OF

ILEAL PERFORATION

AMONG HOLLOW VISCUS PERFORATION"

During his course in M. S. GENERAL SURGERY from

May 2011 to April 2014 at

Government Kilpauk Medical College & Hospital, Chennai.

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The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on incidence and outcome of ileal perforation" – For Dissertation Purpose submitted by Dr.M.Saravanan, MS (GS), PG Student, KMC, Chennai-10.

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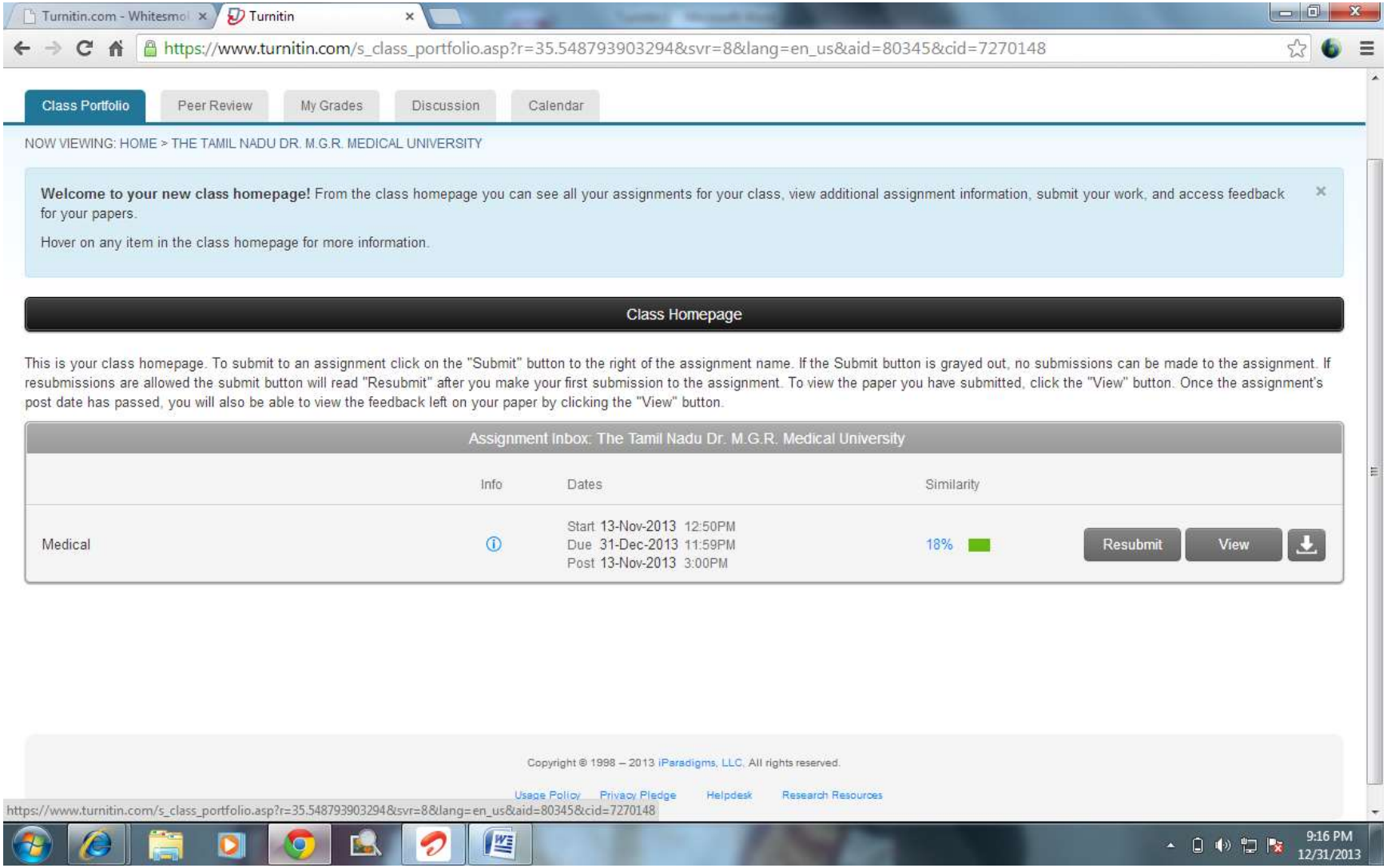
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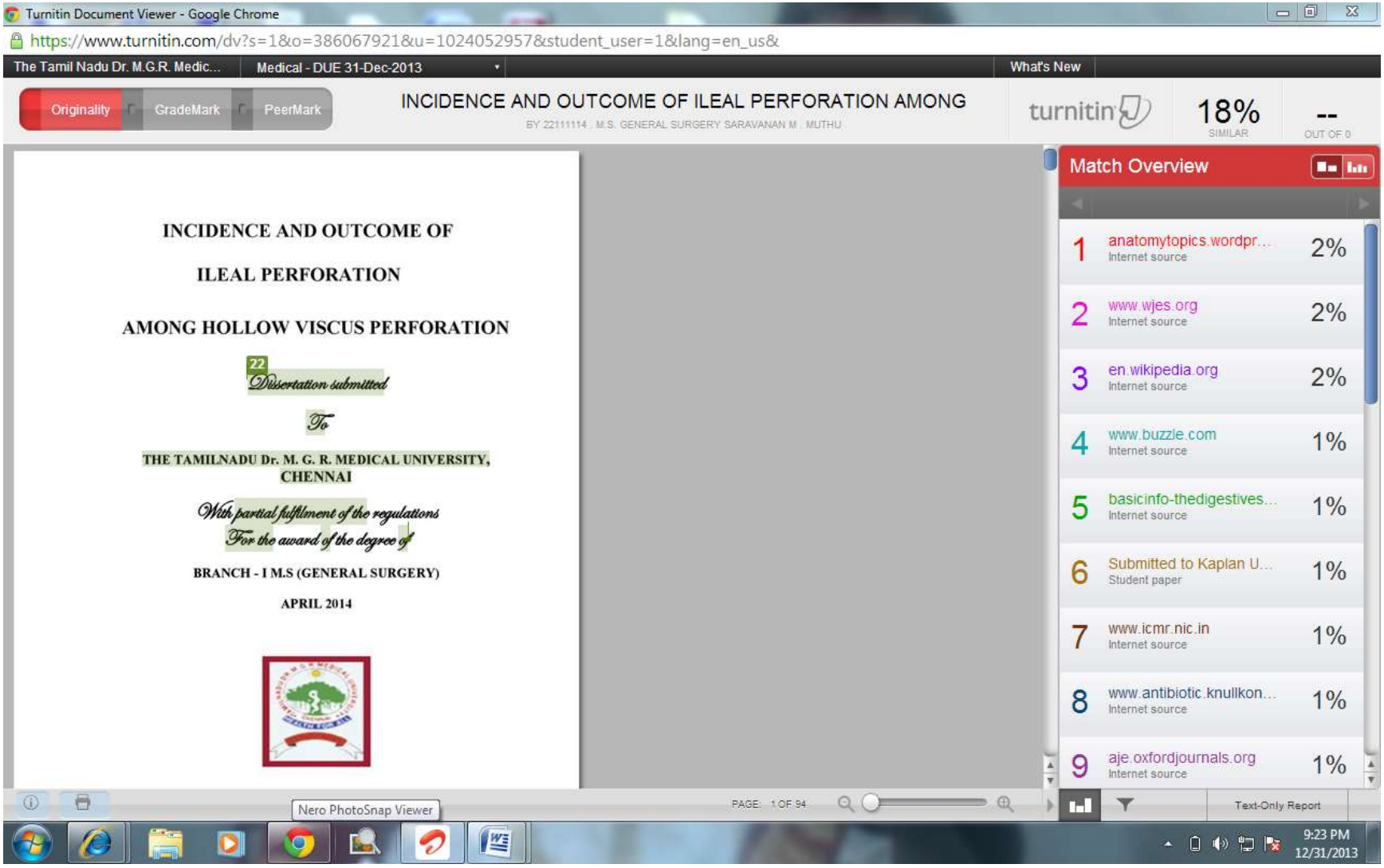
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CONTENTS

ABSTRACT

TITLE:incidence and outcome of ileal perforation among hollow viscus perforation

KEY WORDS: incidence, ileum, viscus., perforation, typhoid, duodenum,jejunum .

Introduction : World wide hollow viscus perforation was common emergency problem in surgical casualty department . incidence of hollow viscus perfartion is variable from regional wise statistics. But all over worldwide Male incidence was greater than female incidence.

male:female ratio was 4:1.

In my study sex ratio out come is 3.5:1. Life style modification , stress free life regularized food habit all will be a deciding factor for hollow viscus perforation

DISCUSSION: A study was conducted in Govt Kilpauk medical College Hospital **and** Govt royapettah hospital by assessing the 60 cases of hollow viscus perforation .Among sixty patient 14 patient was female and 46 patient was male.Patient are included in this study based on clinical and radiological based evidence.Patient taken for emergency laparotomy procedure , closing of perforation by Primary closure ,

resection and anastomosis, patch closure.postoperatively patient kept in Sicu and surgical post operative ward .

Post operative followup based on regarding day of oral feeds started , surgical wound infection, wound gaping and those patient undergone for secondary suturing.Among ileal perforation 8/18 patient wound became infected .5/8 infected ileal perforation wound undergone secondary suturing.

Those patient undergone for secondary suturing their DALY (daily adjusted life year) was more. Data analysed regarding food habits of every patient .

Those patient had an hotel food habits , smoking, alcoholism, stress ful life they became as victim for hollow viscus perforation.

The common age groups are 3^{rd} and 4^{th} decades. Among sixty patient 24 patient belongs to 3^{rd} and 4^{th} decades. Ileal perforation due to typhoid is an important etiological factor at 3^{rd} and fourth week of infection .Widal test and HPE is an important test to rule out other etiological factors of perforation.

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INTRODUCTION

Free perforation of a duodenal or gastric ulcer into the peritoneal cavity can be catastrophic, life threatening event. Common risk factors are NSAID usage, elderly patient, smoking, etc. Smoking causes cocaine induced vasoconstriction, leading to ischemia which can cause perforated ulcer of the pre-pyloric antrum.

Upto 10% of perforation is accompanied by haemorrhage. In duodenum, perforation involves anterior wall: In gastric ulcer, it involves lesser curvature.

There are three clinical stages of free perforation to complete evolution of peritonitis. Initial symptoms are caused by the sudden outpouring of caustic gastric juice into peritoneal cavity. The second stage is caused by neutralization with gastric juice, and the last one is frank peritonitis which may end up with death.

Ileal perforation is still confusion and controversy over the diagnosis and optimal surgical treatment of non traumatic terminal ileal perforation-a cause of obscure peritonitis.

This study is an observation study aimed at evaluating the clinical profile, etiology and outcome of patients with non-traumatic and traumatic ileal perforation.

AIM OF THE STUDY

Among small intestine perforation, the ileal perforation is the major root cause for faecal peritonitis that may lead to major wound infection, wound gaping that necessitate secondary suturing. Like appropriate measures are not taken timely patient may die due to septic shock.

The major morbidity and mortality will be prevented by time being measures such as appropriate antibiotic, fluid management and resuscitation.

In our institute 33% of ileal perforations are reported among

hollow viscus perforation.

The main aim of this study would be to identify:

*) various risk factors of ileal perforation,

*) outcome of ileal perforation among hollow viscus perforation

REVIEW OF LITERATURE

FUNCTIONAL ANATOMY AND PHYSIOLOGY OF STOMACH

The average size of the stomach varies, it contain 1-2 liters of food , liquid during a meal. Normally it stores the food for about 1-2 hours. Always the pyloric sphincter is normally closed. That will help to retain the food and stomach secretions within the stomach. After the process the chyme is formed ready to leave the stomach. Pyloric sphincter become relax and allow a small amount of chyme to pass into the duodenum by the process of ***gastric emptying***.

ANATOMY OF STOMACH [1]

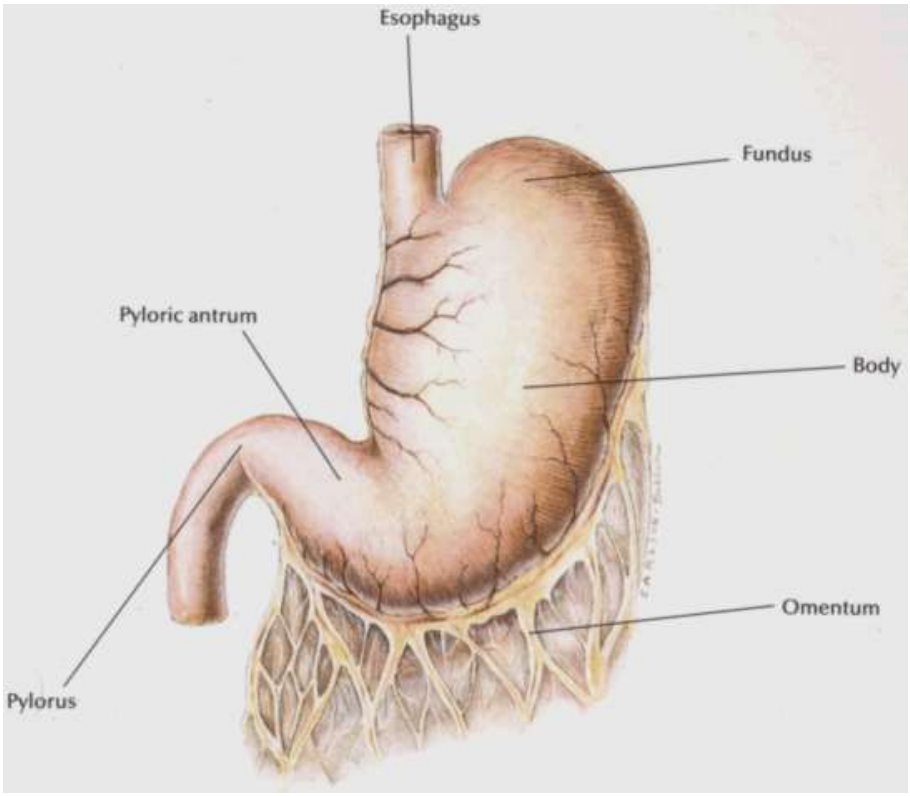


Fig 1: Anatomy of stomach

CELIAC ARTERY BLOOD SUPPLY OF STOMACH

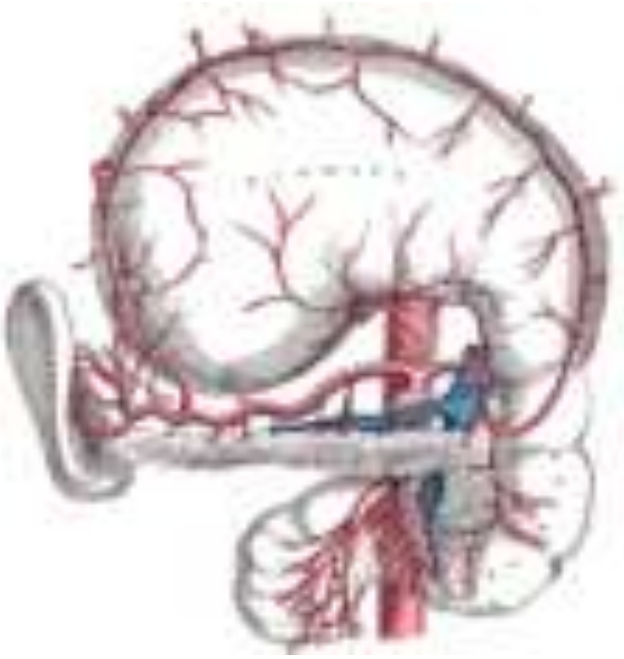


Fig 2: Celiac artery blood supply of stomach

Stomach receives its blood supply from

*) short gastric artery – a branch from splenic artery, which in-turn is a branch of celiac trunk

*) Left gastro-epiploic artery – a another branch from splenic artery;

*) left gastric artery – this is an direct branch of celiac trunk and

*) right gasric artery – a branch from hepatic artery, which in-turn is a branch of celiac trunk, via the right gastro-epiploic artery.

SMALL INTESTINE

The small intestine is the longest section of the digestive tract and consists of three segments which connecting a passage from the pylorus to the large intestine.

There are three parts of small intestine:

1. duodenum

2. jejunum and

3. ileum

The length of duodenum is 25 cm. which is divided into four parts:

D1: 5 cm length,

D2: 7.5 cm length,

D3: 10 cm length

D4: 2.5 cm length

Duodenum is 5 cm in diameter and retro peritoneal structure ,it is the most fixed portion . finally, it joins the jejunum - the second portion of smallintestine.

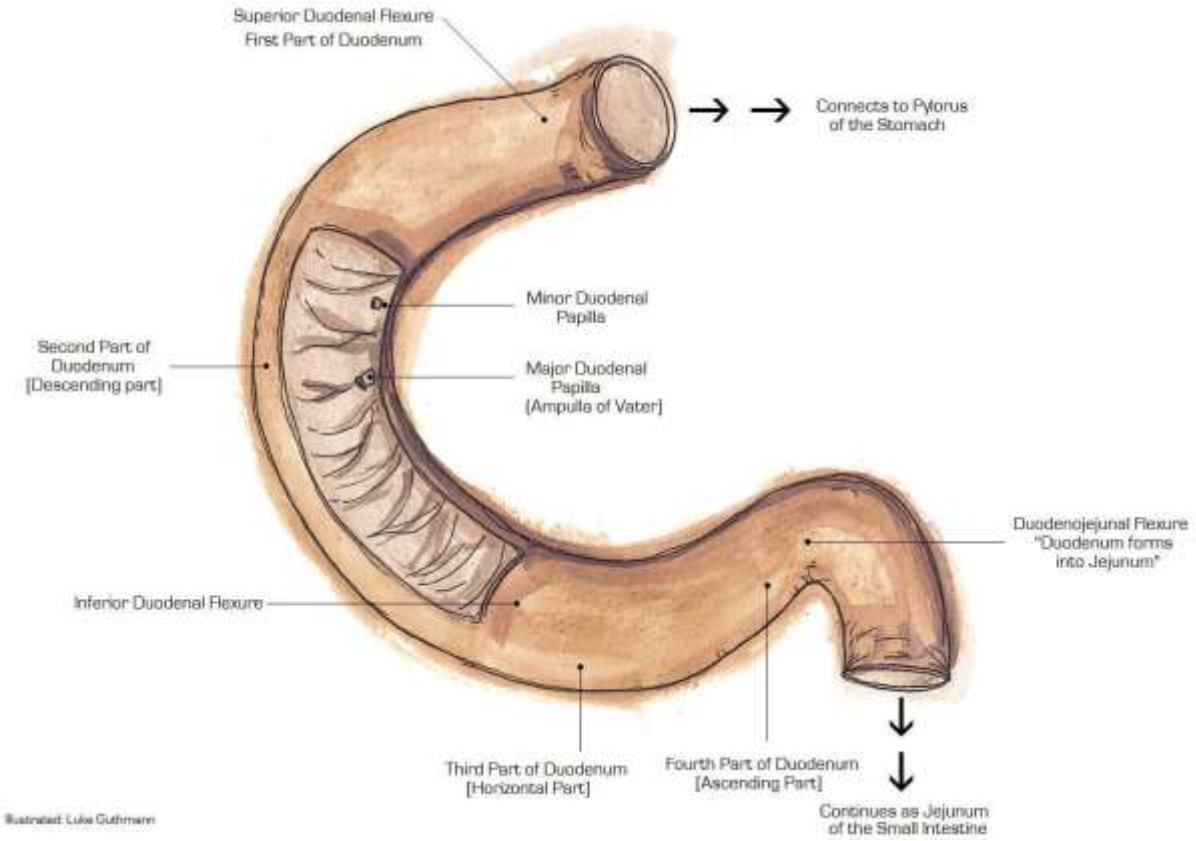


Fig 3: Parts of Duodenum

1st PART:

The first part of duodenum measures around 5cm in length. The first (superior) part begins as a continuation of the duodenal end of the pylorus. It is the only intra-peritoneal portion of the duodenum.

2nd PART:

This is three inches length. The second also known as descending part of the which begins at the superior duodenal flexure. The pancreatic duct and common bile duct enter the descending duodenum, commonly known together as the **hepatopancreatic duct** by the major duodenal papilla which is called asAmpulla of Vater. The 2nd part of the duodenum preserve the opening for minor duodenal papilla, which is an entrance for the accessory pancreatic duct .Also called as duct of santorini.The embryological junction between the foregut and mid-gut lies just below the major duodenal papilla.

3rd PART DUODENUM :

This is also known as inferior or horizontal part of the duodenum which is begins at the inferior duodenal flexure and passes transversely to the left . It crossing the right ureter, right testicular and vertebral column from right to left

4th PART:

The fourth part of duodenum is 5cm long. The course of 4th part is run upward and to the left to the duodenojejunal flexure. This flexure is kept in position by a, the ligament is known as ligament of Treitz.

The 4th part is posteriorly related with :

1. aorta's left margin

2. left psoas muscle medial border

DUODENUM -MICRO ANATOMY

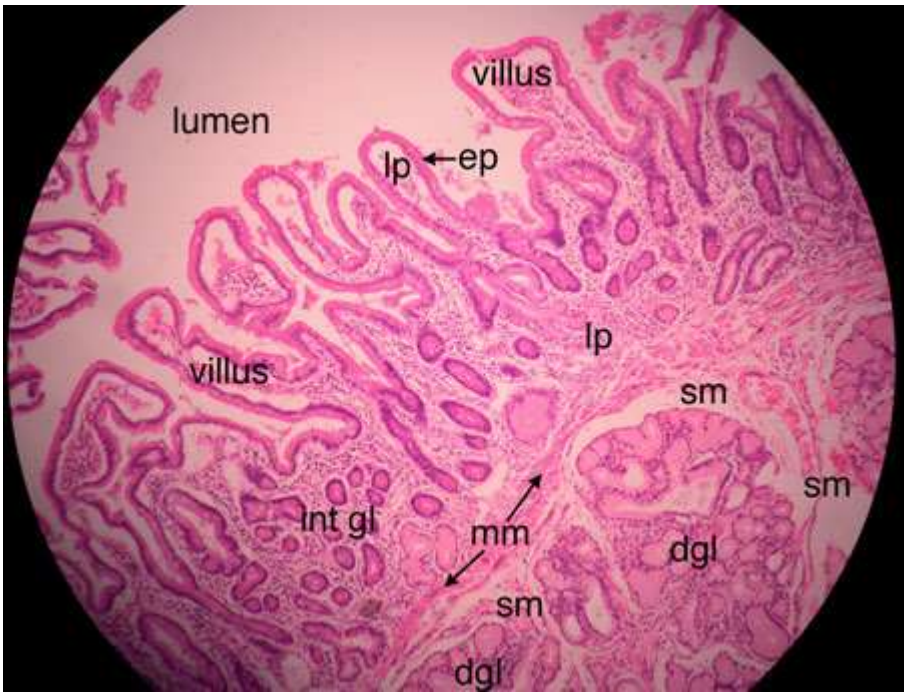


Fig 4: Micro-anatomy of Duodenum

Ep - epithelium Mm - muscularis mucosa \

Dgl - brunner's gland Sm - submucosa

Int gl - interstitial gland

FUNCTIONS OF DUODENUM

After the gastric emptying the duodenum receives the acidic chyme from the stomach after pyloric sphincter relaxation,

1.The duodenal epithelium release two hormones, named as **secretin and cholecystokinin**.

2 . The first enzyme**, Secretin** is released due to excess acids in the duodenum, the other enzyme named as **cholecystokinin** is stimulated by the presence of amino acids and fatty acids.

3.The function of **secretin** stimulates the gallbladder and release the alkaline bile.The duodenum , participate in regulating the rate of gastric emptying, stimulate the hunger signals.

4. The duodenal crypts are increasing the surface area of the intestinal membrane, and ensure the better digestion and absortion.

5. The Brunner's glands exclusively present in duodenum .The function of Brunner's gland are producing a mucus-rich alkaline secretion.The main advantage of alkaline secretion was it protects

the duodenum from the acidic chyme, and lubricates the intestinal wall.

6. Brunner's glands are also concerned with secreting urogastrone, which prevents the secretion of acid and other digestive enzymes by the parietal and chief cells of the stomach.

A GROSS ANATOMY OF DUODENUM AND JEJUNUM

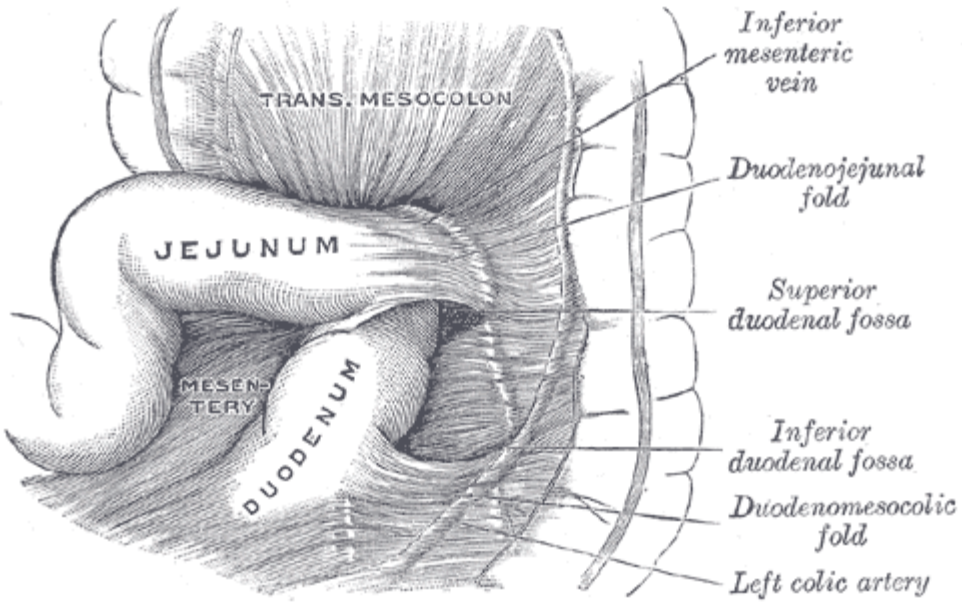


Fig 5: Gross Anatomy of Duodenum & Jejunum

JEJUNUM

It is the longest part of the small intestine and is highly coiled [2]. It has digestive and absorptive functions. Jejunum occupies the ventral part of the abdominal cavity, filling those parts that are not occupied by other viscera. It lies on the abdominal floor, separated from the parietal peritoneum by the greater omentum. This conveys the blood vessels and nerves and houses lymph nodes.

The mesentery converges to its root. This is where the cranial mesenteric artery branches off from the aorta.

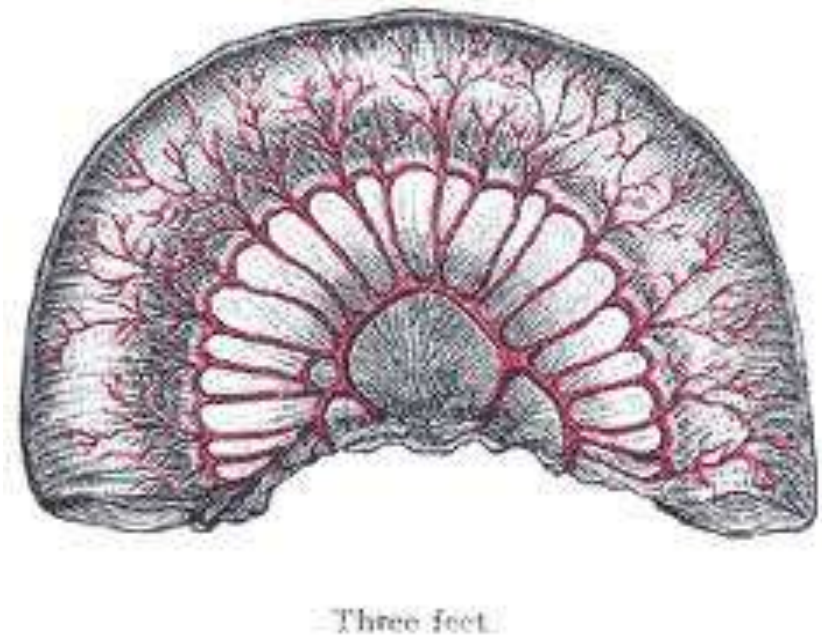


Fig 6: a picture showing blood supply of jejunum

Histology of jejunum

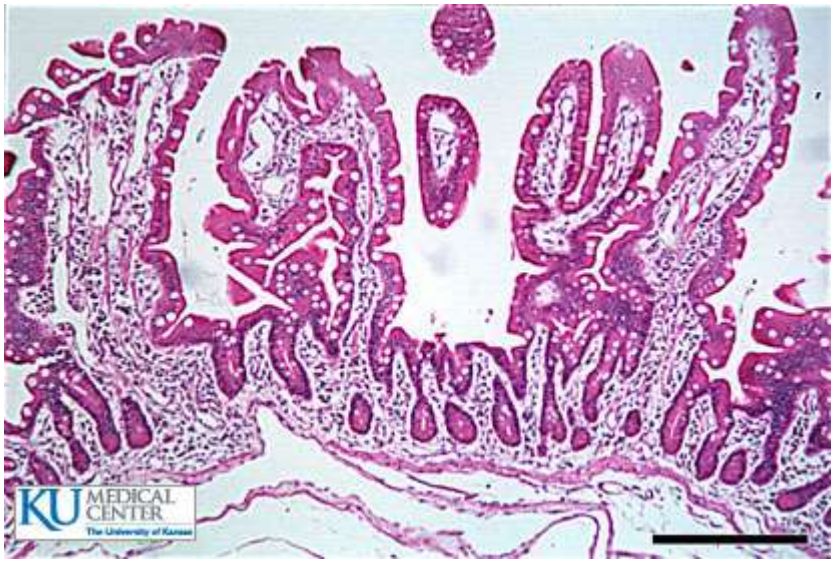


Fig 7: Histology of Jejunum

In jejunum, the villi are long and narrow.

In duodenum, the villi are broad and short.

JEJUNAL PERFORATION

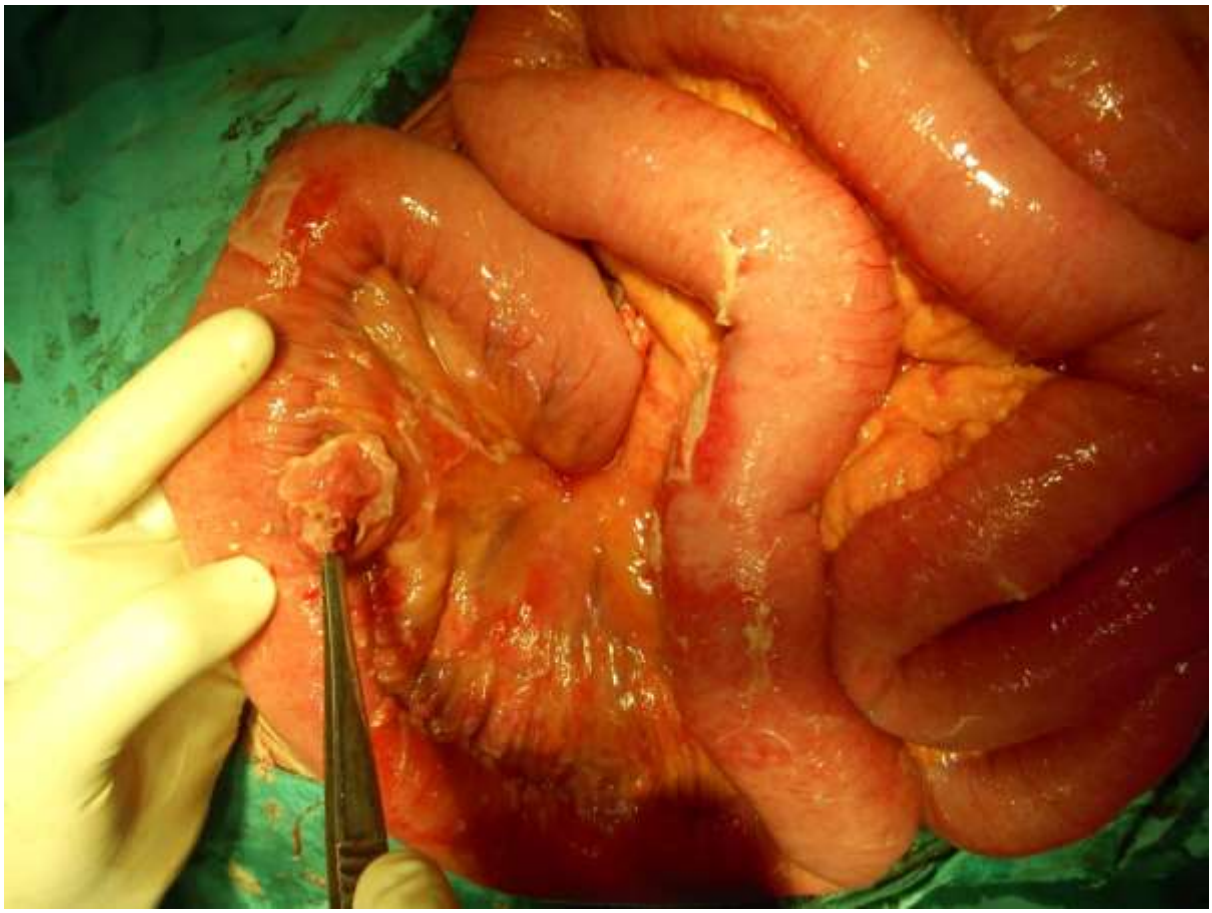


Fig 8: A jejunum with perforated diverticulum

(A Complication of jejunal diverticulum)

The histology of small intestine

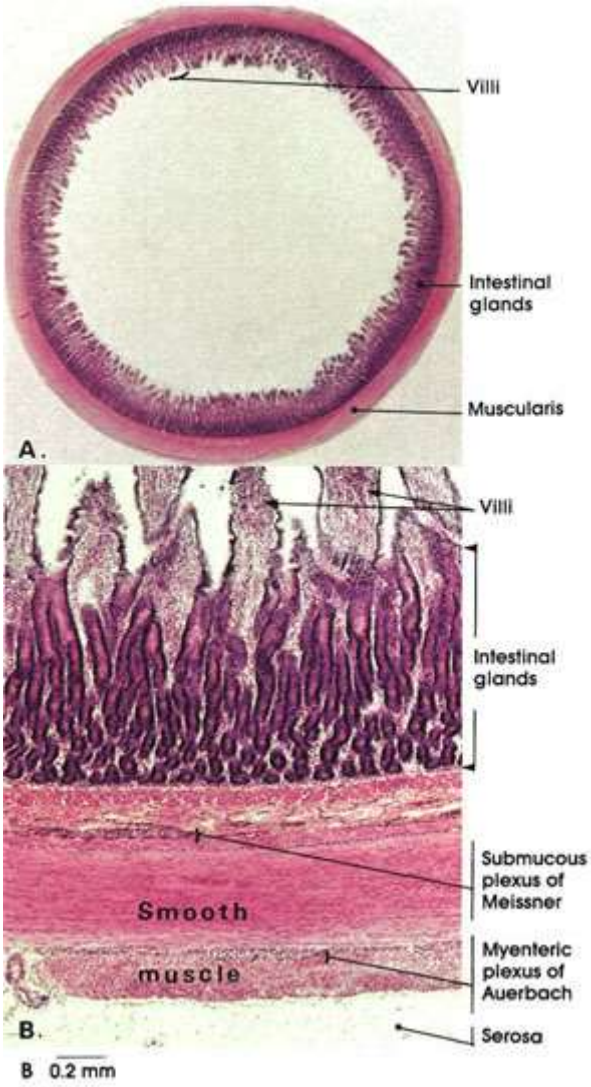


Fig 9: Histology of small intestine

The jejunum contains the following layer:

Mucosa

Submucosa

muscular layer

serosal layer

The lamina propria is present in submucosal layer

Peyer’s patch absent in jejunal wall

In jejunum villi are long and tall

Jejunum is a common site for non specific perforation which is associated with diverticula and divericulitis. Jejunum connects the duodenum with ileum. It always receives the blood supply from superior mesenteric artery.

FUNCTIONS OF JEJUNUM

The main function of jejunum is absorption. The jejunum has specialised lining, its main function is absorbtion. The enterocyte of jejunum absorb the small nutrient particles which have been previously digested by enzymes in the small intestine. After the absorbtion, nutrients pass from the enterocytes into the enterohepatic circulation .The fat is not absorbed by enterocyte which is enter the circulation through lymphatics

The main arterial supply: Jejunal artery

The main vein supply: jejunal vein

Nerve supply: celiac, vagus

ILEUM

Ileum is an latin word which mean flank, of abdomen wall. Ileum is terminal portion of small intestine .it connect the jejunum with caecum .

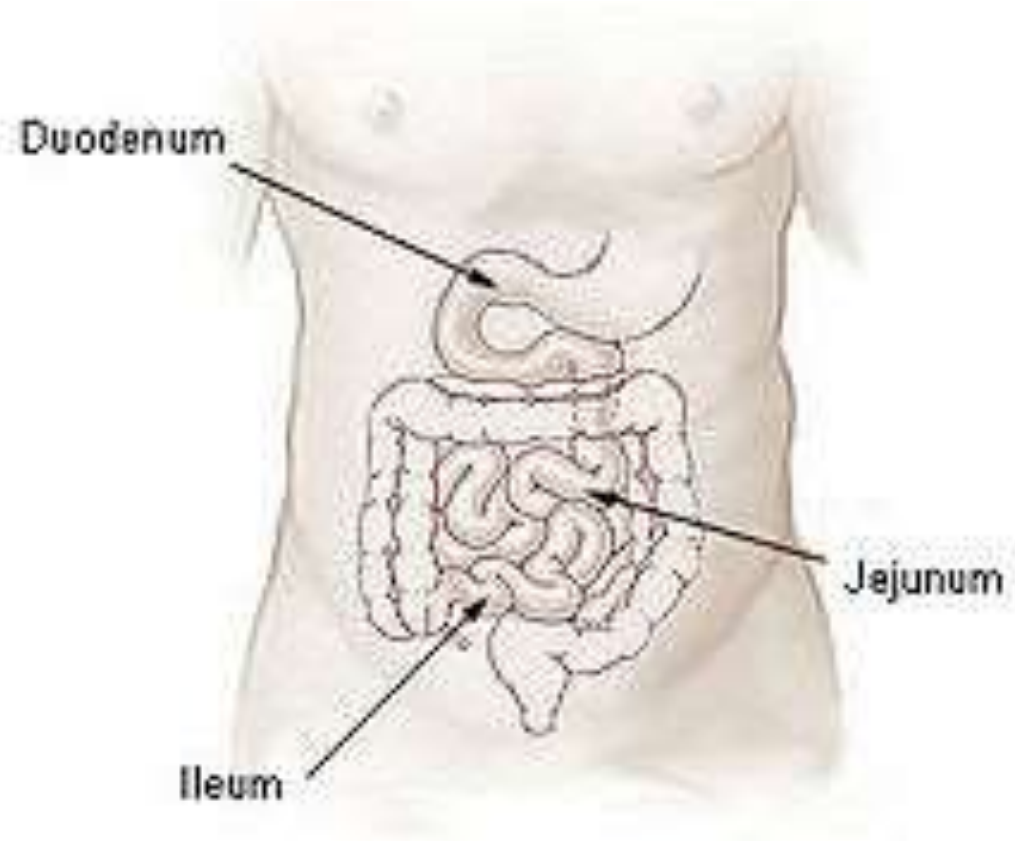


Fig 10: Different regions of small intestine

Caecum is starting point of large intestine. The ileum end up with caecum by ileo caecal valve.

DEVELOPMENT OF ILEUM

In embryo the terminal ileum is connected to the anterior abdominal wall by the structure known as vitteline duct.

ILEAL ARTERIAL SUPPLY

Gets blood from superior mesenteric artery.

The **ileal arteries** feed oxygenated blood into the ileum, a portion of the small intestine.

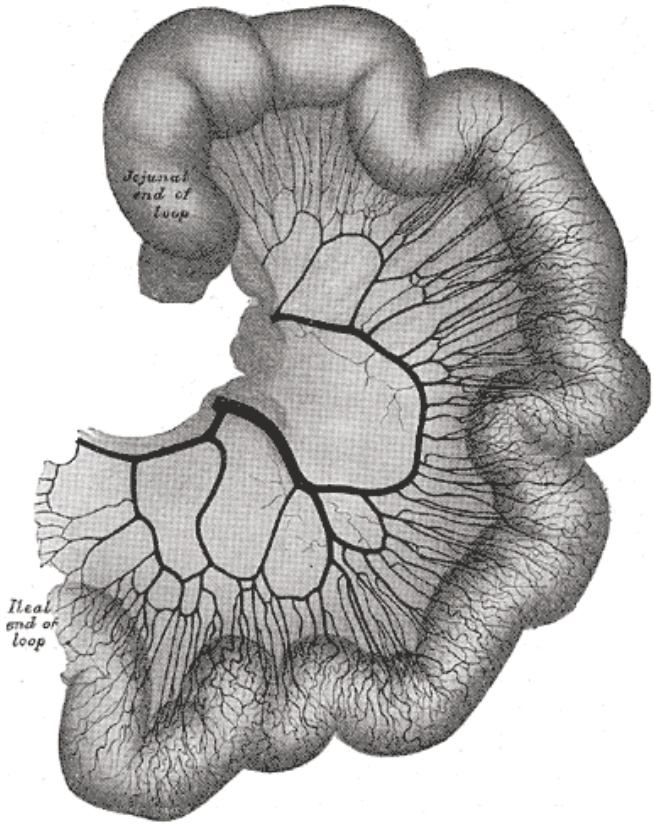


Fig 11: Blood supply of ileum

ILEAL VENOUS DRAINAGE

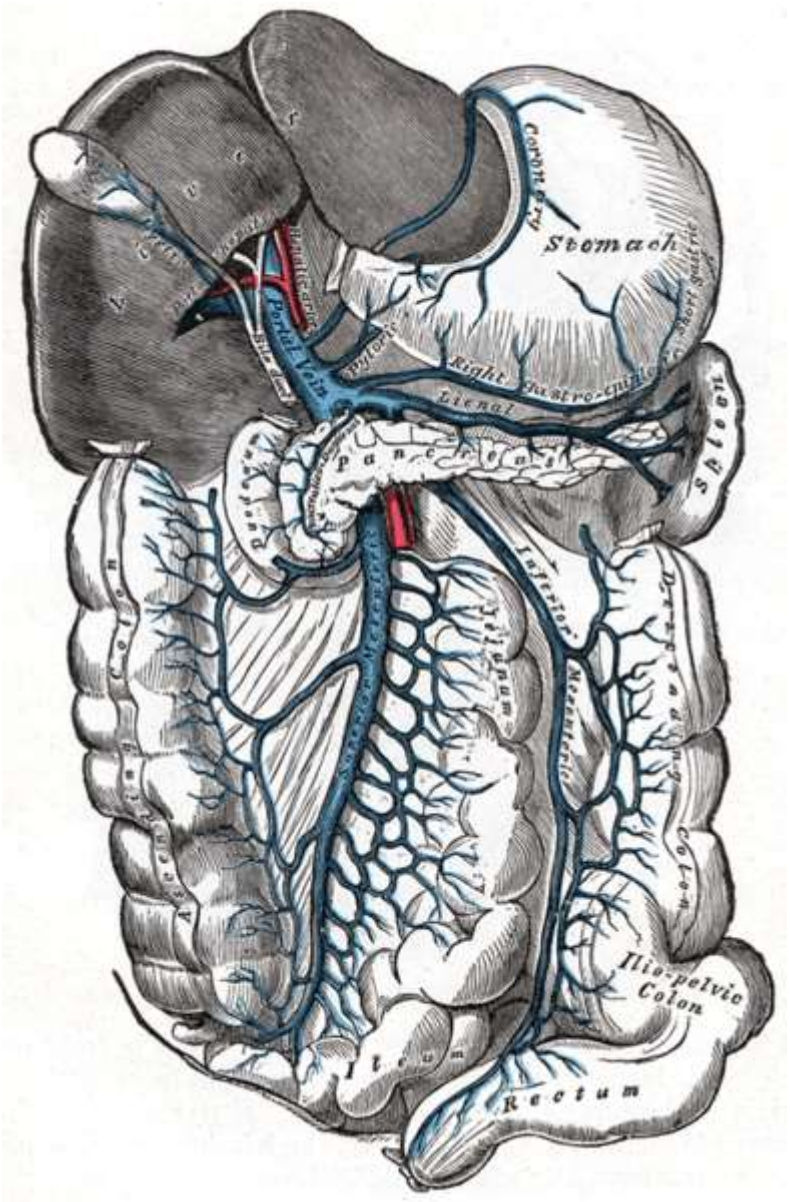


Fig 12: Venous drainage of ileum

The following are Tributaries of the superior mesenteric vein .That will drain the stomach, small intestine, large intestine,pancreas and appendix by :

. right gastro-epiploic vein

- inferior pancreaticoduodenal veins
- veins from jejunum
- veins from ileum
- middle colic vein - drains the transverse colon
- right colic vein - drains the ascending colon
- Ileo- colic vein

HISTOLOGY OF ILEUM

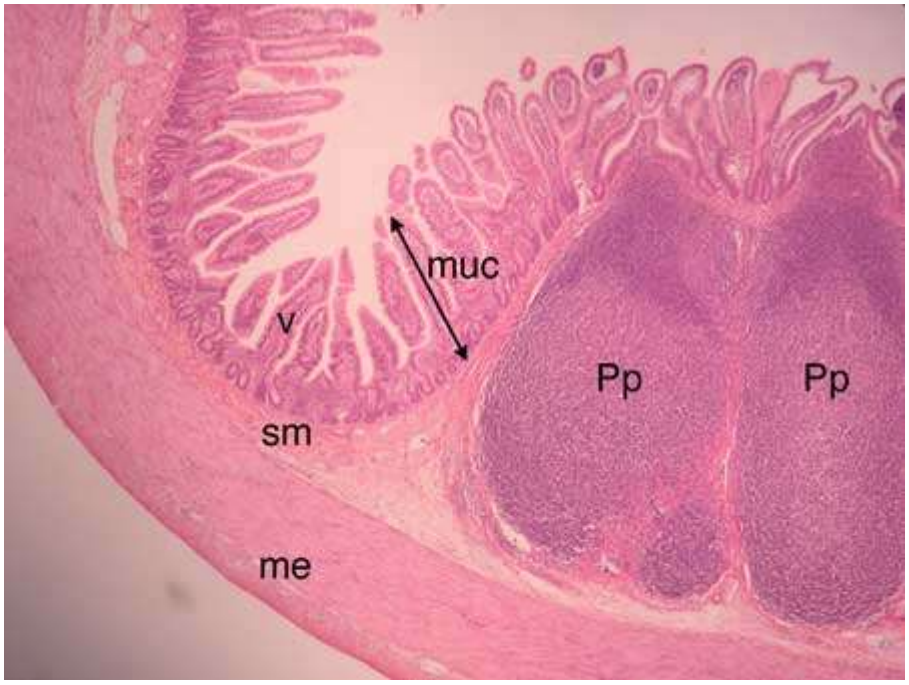


Fig 13: Histology of ileum

Peyers patch is very characteristics of ileum.

Peyer’s patches absent in duodenum and jejunum.

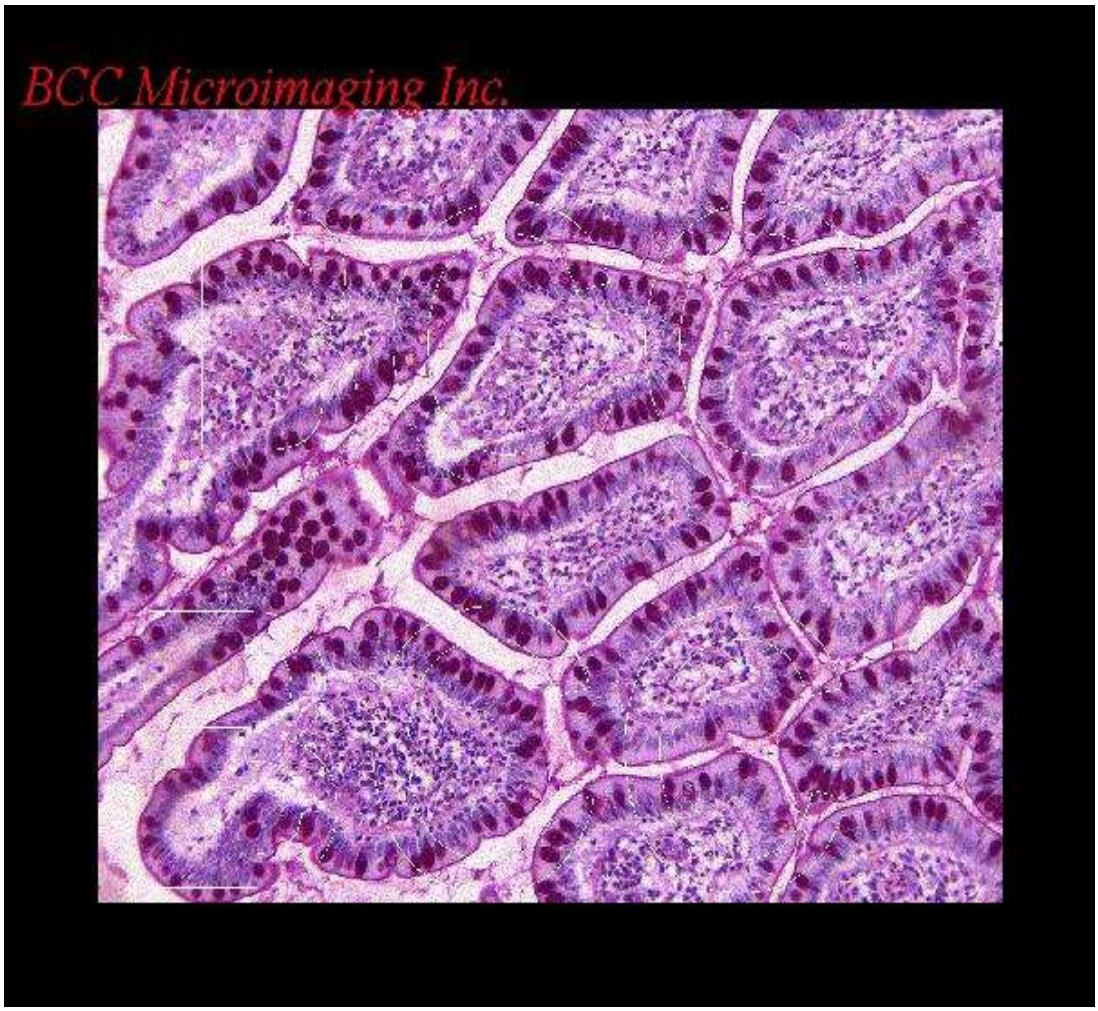


Fig 14: Ileum with lymphoid follicles

Aggregation of lymphoid follicles is characteristic of ileum. It acts as immune defence mechanism.

FUNCTIONS OF ILEUM

The villi present in ileum are large numbers of capillaries that take the amino acids and glucose produced by digestion to the Hepatic vein and the liver. They absorb fatty acids and glycerol and to absorb vitamin B12 and bile salts.

The Diffuse Neuroendocrine System cells (also known as DNES)of the ileum secrete the following hormones, namely.

1. Gastrin,

2. secretin which role in gastric secretion,

3. cholecystokinin act as GB contruction

These are participate in entero-hepatic circulation

The inner lining of the ileum secrete the following enzymes Protease and carbohydrates responsible for the final stages of protein and carbohydrates.

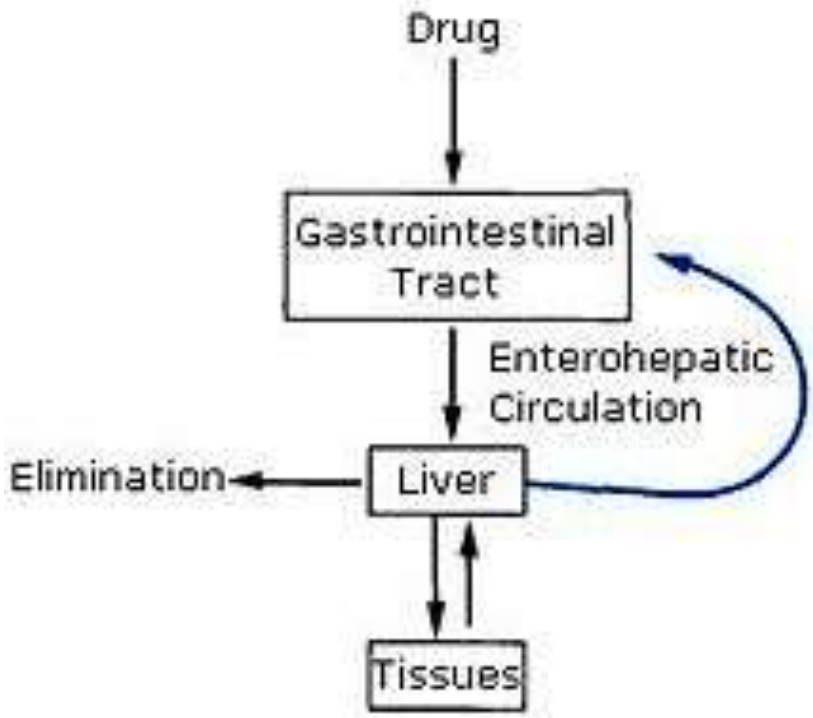


Fig 15: entero-hepatic circulation

1. Jejunum it is an second part of the small intestine and ileum is the lengthiest and the last part of the small intestine**.** Jejunum responsible for Digestion and secretion of enzymes.ileum participate in absorption of vitamin B ‘complex and coupled bile salts.

2. Mucosa Associated Lymph Tissue is minimal in jejunum,but in Ileum has major amounts of Mucosa Associated Lymph Tissue

ARTERIAL STRUCTURE OF JEJUNUM AND ILEUM

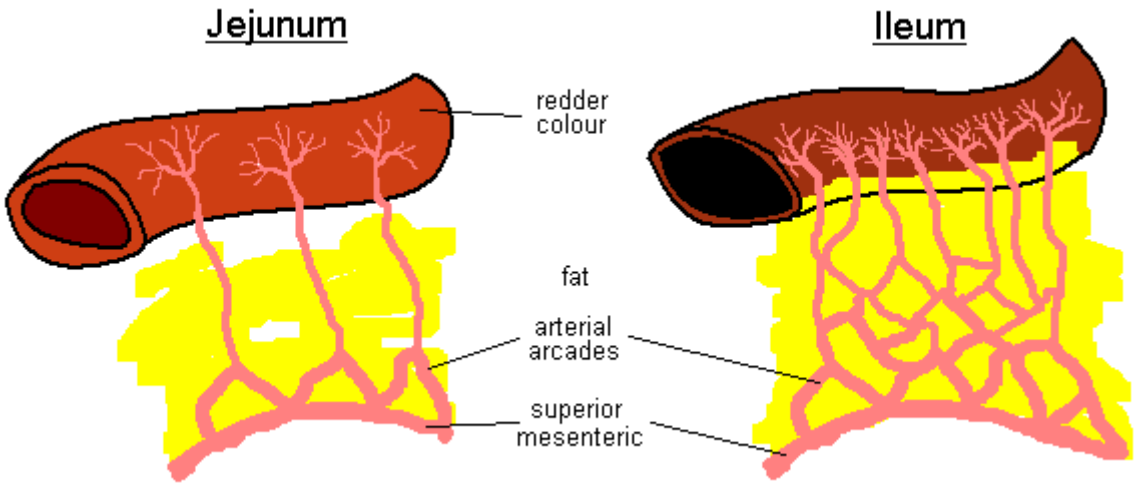


Fig 16: Arterial structure of jejunum and ileum

Area	**Jejunum**	**Ileum**
Location	Jejunum occupy the upper part of intestinal area, by the left side of the transverse colon	Ileum occupy lower And right part of Intestinal area.
Walls And lumen	Wider bored, Thicker walled, and more vascular..	thinner. And less vascular.lumen are narrow

Mesentery	Windows present, fat less abundant	No windows,fat Morevasarecte shorte numerous .
Vessels	Only one or two arcades, with long branches of vasa recta.	Numerous short terminal vessels, after 3 or more arcades
Fat	Fat deposited near the root, and is scanty near intestines.	Fat is deposited throughout.
Peyer's Patches	Absent	present on the ant mesenteric wall.

ANATOMY OF LARGE INTESTINE

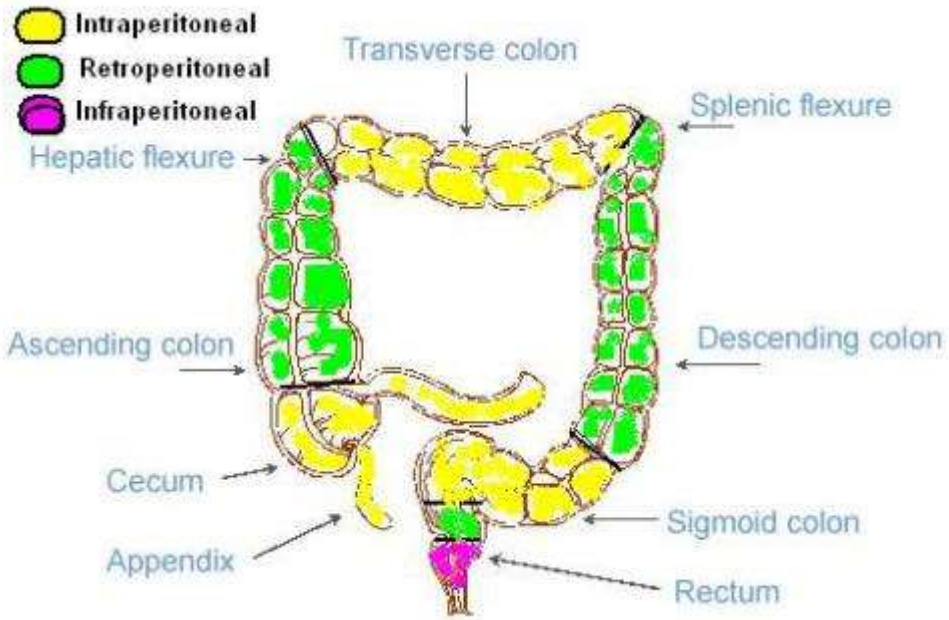


Fig 17: Anatomy of large intestine

Location: occupies a large area in the abdominal cavity, from ileo-caecal junction to anus, approx 1m long.

Distinguishing Characteristics [4, 5]: Has the ***teniae coli***, the three enlargements of outer longitudinal layer of muscularis externa, *haustra* – the bumps formed between each teniae (like little houses), and *omental (epiploic) appendices* – the peritoneum-covered sacs of fat attached all along the colon.

3 tenia are:

- **Omental –** near attachment of greater omentum
- **Mesocolic – @** attachment of mesocolon
- **Tenia Libre (**free)

Blood Supply: Blood upto distal 1/3 of transverse colon is supplied by Superior Mesenteric artery, as it is MIDGUT, after it is HINDGUT, which get blood supply by Inferior Mesenteric artery.

Sup mesenteric artery supplies:

- caecum = iliocaecal a (same as iliocolic a)
- appendix = appendicular br off iliocecal a

ascending colon and proximal 2/3 transverse colon

- iliocecal and right colic a.

Inferior mesenteric artery:

- dist 1/3 transverse colon = middle colic a, left colic a
- desc colon = left colic a, sigmoid a
- sigmoid colon = sigmoid a

The **Ascending Colon** calibre is smaller than the caecum.. The ascending colon commencement at the level of caecum, opposite the colic valve, to the under surface of the right lobe of the liver, on the right of the gall-bladder, where it is lodged in a shallow depression, the **colic impression;** here it bends abruptly forward and to the left, forming the **hepatic flexure**

TRANSEVERSE COLON

The longest and most movable part of the large intestine,It passes from the right hypochondriac region with downward convexity and traverse across the abdomen, opposite to the confines of the epigastric and umbilical zones, into the left hypochondriac region, where it curves sharply on itself beneath the lower end of the spleen, forming **the splenic flexure.**

The **transverse mesocolon** is a structure most completely invested by parietal ,visceral peritoneum. It is connected to the structure inferior border of the pancreas by a duplicature of that membrane**.** The upper surface of the transverse mesocolon related with the liver and gall-bladder.

THE DESCENDING COLON

This is 10 inches long. This is commenced at the level of **splenic flexure which** is situated at the junction of the transverse and descending parts of the large intestine.The major relation are the splenic lower end and pancreatic tail, the flexure is the end of the transverse colon ,usually lies in contact with the front of the descending colon.

The left colonic **splenic flexure** is attached to the diaphragm by peritoneal fold, called as **phreni-colic ligament.**

Lymph Drainage:

Lymph vessels always follows the arteries. Lymph drains into the lymph nodes .The lymphatic draining area named by the arteries which supplies the area.

PREDISPOSING FACTORS OF HOLLOW VISCUS PERFORATION [6]

- peptic ulcer disease which is caused by stress ,smoking alcohol,
- acute diverticulitis -most common site is duodenum
- acute appendicitis
- Inflamed Meckel diverticulum.
- Indeed, acute appendicitis is still one of the common cause.
- The major cause of intestinal perforation in elderly people is unauthorized drug intake : a.Ingestion of aspirin, b. nonsteroidal anti-inflammatory drugs (NSAIDs), c.steroids - Intestinal perforation from such causes is particularly observed in elderly patients
- Bowel injuries associated with endoscopy, ERCP, colonoscopy
- Inflammatory bowel disease
- Perforation secondary superior mesenteric artery occlusion disease (eg, ischemic colitis)
- Small Bowel perforation secondary to intra-abdominal malignancy: lymphoma, metastatic renal carcinoma.

CIGARRETE

NSAIDs

STRESS

DEFENSIVE MECHANISM IN STOMACH:

The stomach has gastric MUCOSAL barrier which it allow the stomach to contain the low Ph acid.

The mucosal barrier consists of three protective components:

A special mucus covering

A compact epithelial cell lining

A special mucus covering, secreted by surface epithelial cells and foveolar cells. The surface epithelial cells secrete the bicarbonate ions which act to neutralize harsh acids.

RISK FACTORS FOR GASTRIC PERFORATION

Peptic ulcer disease:

Other Causes of peptic ulcer disease:

The bacteria causing peptic ulcer disease is

Helicobacter pylori (H. pylori)

nonsteroidal anti Inflammatory drugs

Zollinger-Ellison syndrome

Drink alcohol regularly

> age 50 years old or older

HELICOBACTER PYLORI PATHOGENESIS:

50% of world population infected with H.PYLORI. It may cause chronic gastritis, sequence of inflammation, metaplasia, dysplasia & carcinoma and Gastric lymphoma.

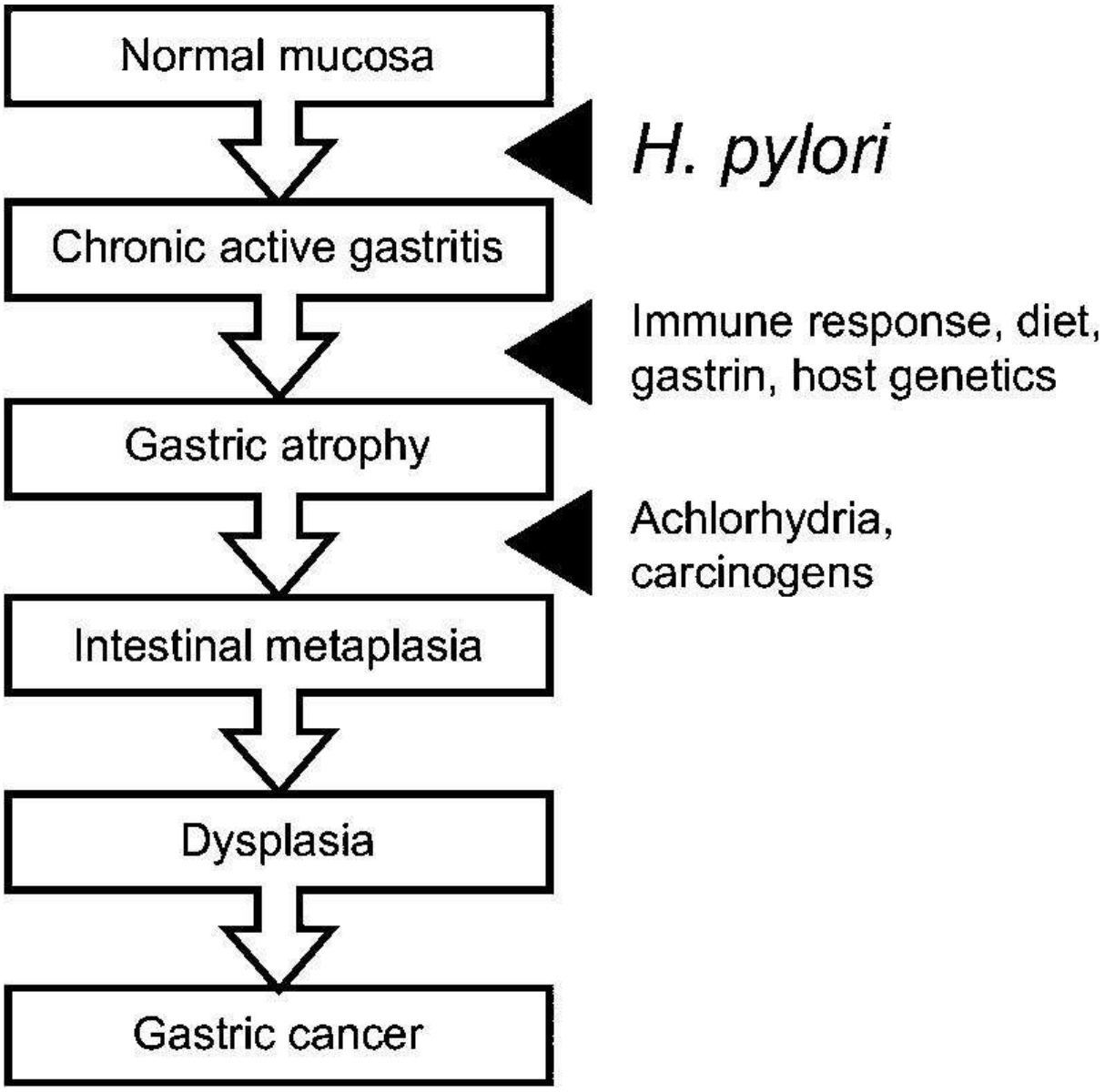


Fig 18: H. pylori pathogenesis

COMPLICATION OF GASTRIC ULCERS:

Bleeding

Perforation

Obstruction

Perforation is the 2nd most complication of gastric ulcer.

What will happen if perforation occurs?

Perforated gastric ulcer cause more mortality than duodenal ulcer because following reasons:

Patient with more advanced age

Increased medical co morbidities

Delay in seeking medical attention

Larger size of gastric ulcer

PERITONITIS

STAGES OF PERITONITIS [7]:

a initial peritonitis

b. neutralization stage

c.terminal stage

Line of management in peritonitis: abdominal decompression by

. ryle’s tube

. intravenous fluids

. intravenous antibiotic

. appropriate surgical management

ILEAL PERFORATION

Causes are:

1. First and foremost cause is enteric fever which responsible for 62% of ileal perforation,
2. 2nd common cause is nonspecific inflammation,it tooks 26% of ileal perforation,
3. Small bowel obstruction(6%),
4. Tuberculosis is foremost cause for multiple ileal perforation(4%) and
5. Radiation enteritis:1%

Typhoid fever is caused by the gram-negative bacillus *Salmonella typhi* serovar. This is a global health problem in poor countries.In developing countries, Regions with contaminated water supplies and inadequate waste disposal are major root cause for a high incidence of typhoid fever.

The most life threatening complications of typhoid fever are intestinal bleeding and ileal perforations, the main pathogenesis behind this complications are necrosis of Peyer's patches in the terminal ileum.

The majority of typhoid fever patients who develop perforation at the 2 -3 weeks of after the onset of illness.

.The mechanism of intestinal perforation in typhoid fever is hyperplasia and necrosis of Peyer's patches of the terminal ileum. The lymphoid aggregates of Peyer's patches extend from The lamina propria to the submucosa, so that in the presence of hyperplasia the distance from the luminal epithelium to the serosa is bridged by lymphoid tissue.The residing sites of *S*. Typhi is within mononuclear phagocytes of Peyer's patches of ileum.

ILEAL PERFORATION

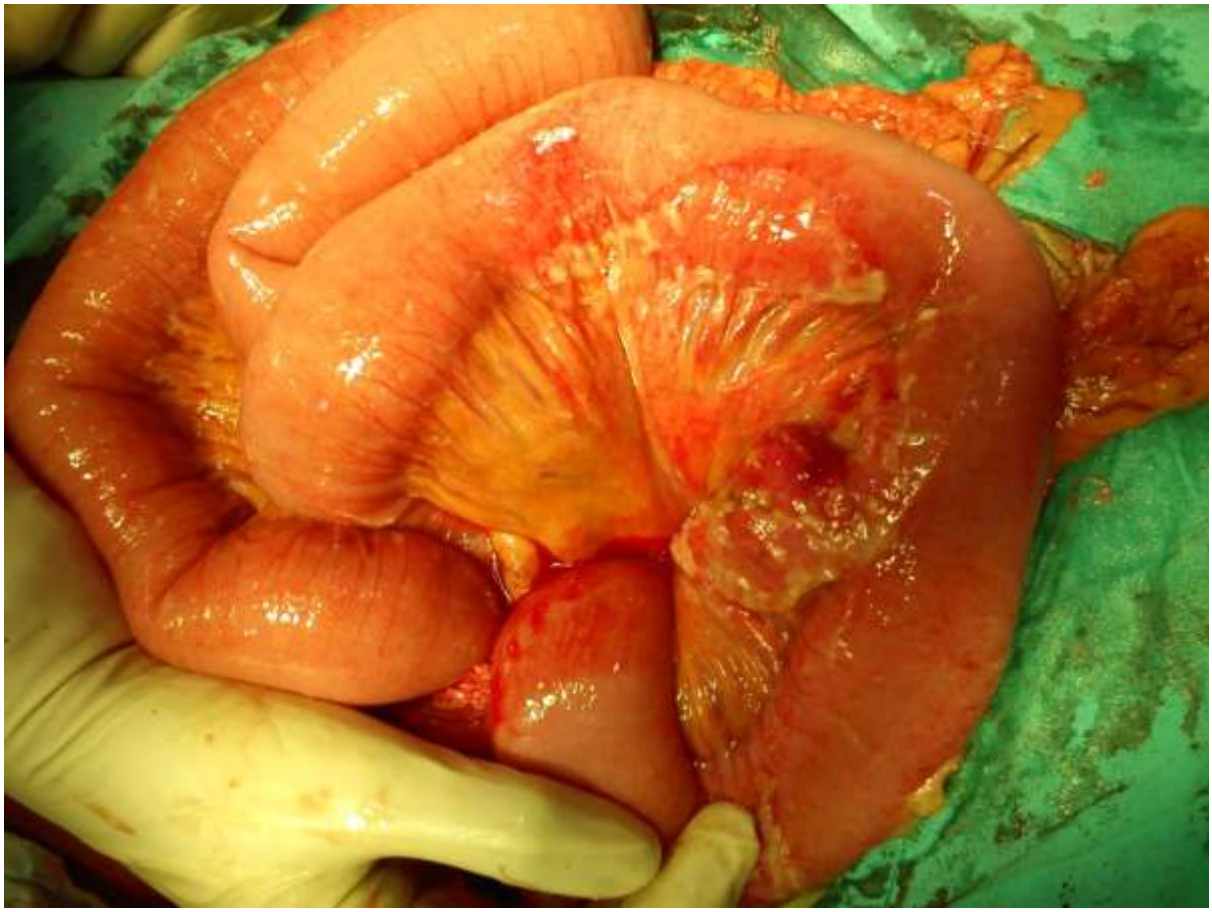


Fig 19: Typhoidal perforation

Due to multiplication of organism occurs within the Peyer's patches , resulting in ulceration, bleeding, necrosis, and, in extreme cases, full-thickness perforation may be occur.

CLINICAL FEATURES:

Male incidence greater than female 5:1

Common age group 4th decade

Abdominal pain

Vomiting

Fever

Loose stools

SIGNS:

Tenderness

Guarding

Rigidity

Distention

Investigatigation

Blood count shows leucocytosis

Leucopenia in some patients

x-ray shows gas under diaphragm

USG SHOWS: free fluid

CT scan candiagnose even minimal free fluids & minimal amount of air up to 5ml.

MANAGEMENT:

Simple debridement,

repair of perforation is shown to be effective,

resecting 10 cms on sides of proximal and distal perforation.&

anastomosis of both ends

appropriate antibiotics depending upon blood culture and

sensitivity

COMPLICATION OF ILEAL PERFORATION:

1. peritonitis

2. The most common postoperative complication:

wound infection[18]

enterocutaneous fistula

mortality

The complications significantly increase the hospital stay and

DALY.

DALY is disability adjusted life year of human in his whole life

Priod .Because of secondary infection DALY IS

Significantly increased in association with ileal perforation

ENTEROCUTANEOUS FISTULA: One of the treaded complication of ileal perforation is enterocutaneous fistula which is commonly associated with malnutrition, immunocompramised state ,older age group

ILEAL PERFORATION NON SPECIFIC TYPE

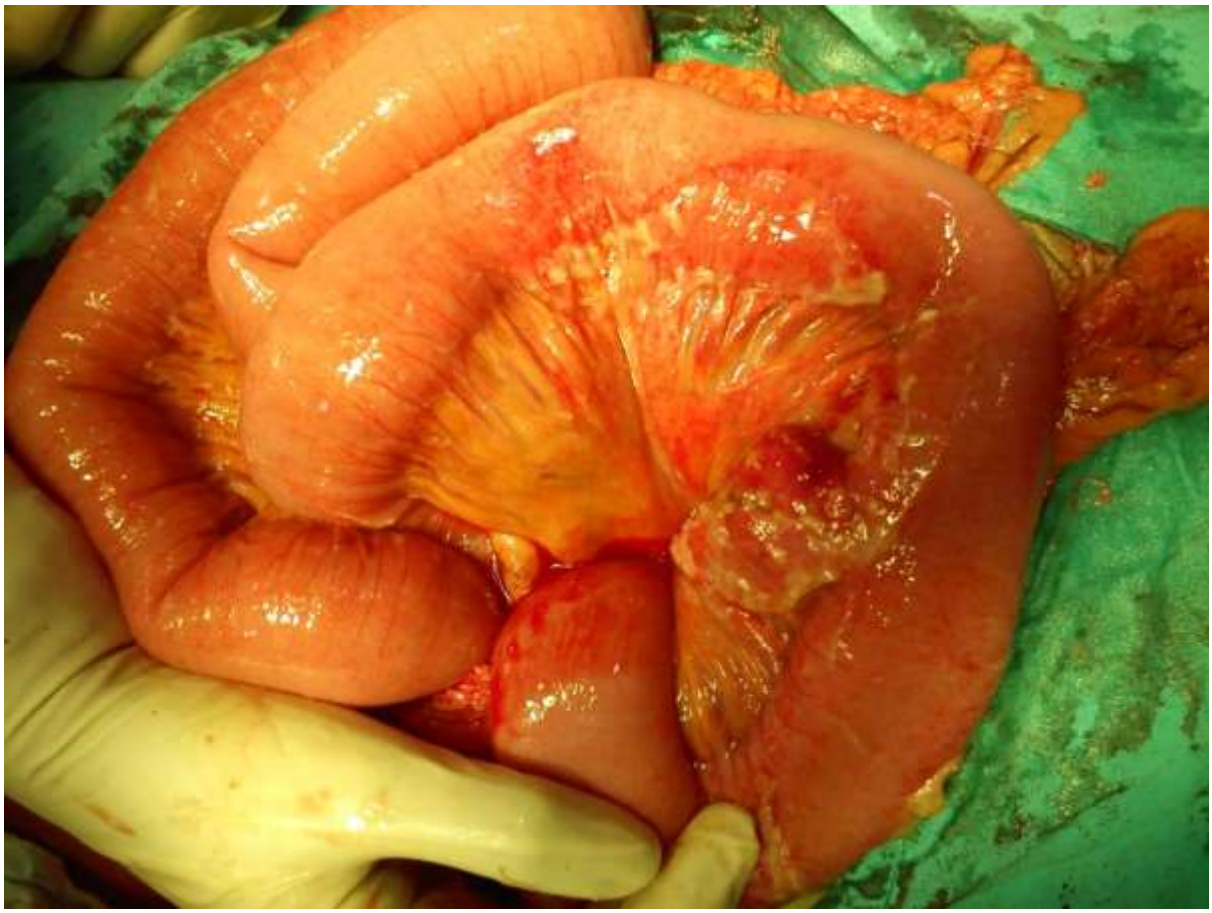


Fig 20: Non-Specific Ileal Perforation

MANAGEMENT OF ENTEROCUTANEOUS FISTULA

Investigation: The aim of investigations are

stabilization of patient

Adequate nutrition

Allow Ileal perforation fistulas to get matured

investigation to locate the fistula

1.Adequate hydration

2.drainage of sepsis

3Adequate nutrition

4.control of fistula drainage

5.adequate skin care

6.Surgical closure of fistula at 4-6 weeks

Spontaneous closure of fistula allowed only for following circumstances

a.fistula tract <2c.m

b. fistula arise from jejunum ileum at the ligaments of trietz

Small bowel fistulas may take at least 40 to 60 days for spontaneous fistula

RADIATION INDUCED ILEAL PERFORATION

INTRODUCTION: The undesired complication of radiation therapy is radiation induced enteritis.

It divided into two components:

Acute and chronic

75% acute radiation enteritis is transient condition

5-15% radiation enteritis is chronic type

PATHOPHYSIOLOGY: The principal mechanism of radiation induced cell death is believed to be apoptosis resulting from free-radicals induced breaks in double stranded DNA.

Required dose of radiation administered injury **is 4500cGy**.

Clinical features:

Nausea

Vomiting

diarrhoea and

abdominal pain.

If perforation occur features suggestive of acute abdomen will be occurred. Terminal ileum is the most affected segment.

Other complication of chronic radiation enteritis was intestinal obstruction.

TREATMENT:

supportive therapy for dehydration

Surgery is indicated in:

perforation,

Obstruction

Intra abdominal abscess

fistula

outcome: surgery with chronic radiation associated with high morbidity and mortality

CROHNS DISEASE

Crohn’s disease is a type of inflammatory bowel disease ,it may affect any part of the Gastero.Intestinal Tract from mouth to anus [3]. Males and females are equally affected.

The Smoking is an important risk factor to develop Crohn's disease than non-smokers. The Crohn’s disease can occur at any age. The common age group occurs in Crohn's disease is in the teens and twenties, the another peak age incidence is in the fifties to seventies,. The main Treatment options are controlling symptoms.

CAUSES:

The CROHN’S disease is caused by 1. environmental factors and 2.genetic predisposition. It is associated with a frameshift mutation in the NOD2 gene .This is also known as the CARD15 gene point mutations.

The most common overlapping infection for IBD is mycobacterial infections. , .A newer theory hypothesizes that Crohn's results from an impaired innate immunity have suggested a role. The *Mycobacterium avium subspecies paratuberculosis* (MAP), which had a similar pathogenesis

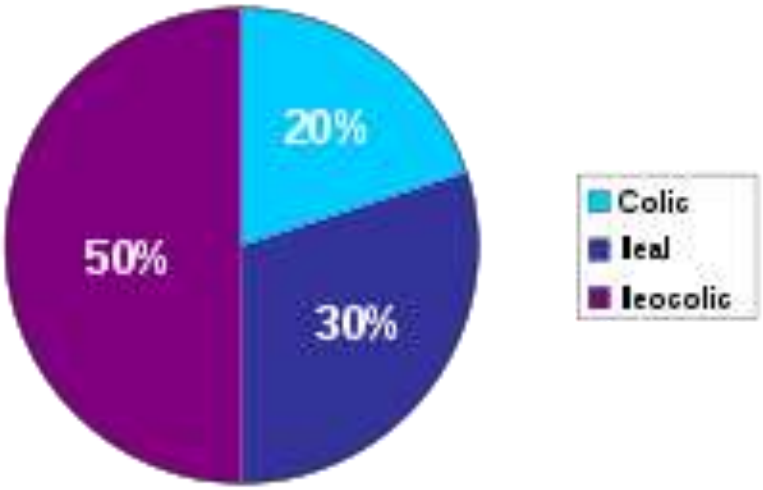


Fig 21: Various sites of Crohn's disease & their incidence

PATHOLOGY

It shows a transmural pattern of inflammation. There is a characteristic sign of CROHN'S DISEASE known as skip lesion is usually an abrupt transition between unaffected tissue and the ulcer Granulomas, are found in 50% of cases of crohn's disease which is most specific pathological feature of crohn's disease.

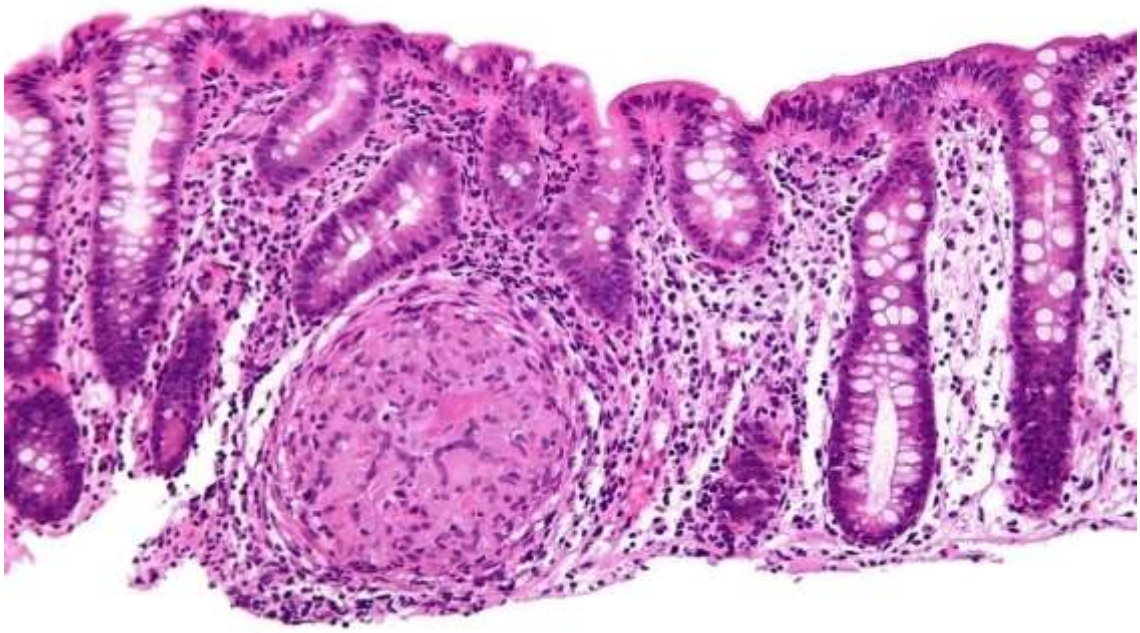


Fig 22: CROHN'S TERMINAL ILEITIS - HISTOLOGY

CLINICAL FEATURES

1.Abdominal pain - initial symptom

2.Bloody diarrhea. Which is typically intermittent in nature.

3.intestinal discomfort due to bloating and flatulence.

4.Intestinal stenosis are also common in Crohn's disease.

5. Itchiness or pain around the anus may be suggestive of inflammation.

Peri anal Crohn's disease: It is associated with

.. FISTULA or abscess around the anal area or anal fissure. .

.Perianal skin tags are also common in Crohn's disease.

. Fecal incontinence may occure .

MANAGEMENT:

5- aminosalicylic acid (5-ASA) formulations, ,

infliximab, adalimumab, certolizumab and natalizumab.

prednisone,

immunomodulators- azathioprine which is the prodrug for 6-mercaptopurine),

methotrexate

severe attacks of Crohn's disease Hydrocortisone can be used ..

Ileal Tuberculosis associated perforation

The ulcerative typeof the intestinal tuberculosis has been recognized as a serious complication of advanced pulmonary tuberculosis. Tuberculosis bacteria entered the G.I tract by haematogenous spread.

Mode of spread:

ingestion of infected sputum.

The other common source are infected contiguous lymph nodes and infected fallopian tube

The most common site of intestinal tuberculosis is ileocaecal region. Intestinal tuberculosis may presents with a **palpable mass** in the right iliac fossa.

Complications :

obstruction,

perforation

mal-absorption especially in the presence of stricture

The **transverse ulcers, fibrosis, thickening and stricturing** of the bowel wall are gross pathologic picture of ileal tuberculosis.

Enlarged and matted mesenteric lymph nodes, omental thickening, and peritoneal tubercles are also other clinical feature of intestinal tuberculosis. In 25% ofextra-pulmonary tuberculosis, Chest X-rays show evidence of concomitant pulmonary lesions..

Other clinical presentations are dysphagia, odynophagia in case of oesophageal tuberculosis, In case of gastro duodenal tuberculosis dyspepsia and gastric outlet obstruction are presenting features**,**

colonic tuberculosis clinical features are Lower abdominal pain and haematochezia .

INVESTIGATION:

following are essential investigation to rule out abdominal tuberculosis ,

1. barium enema,
2. small bowel barium meal

3.other non invasive investigations are

ultrasonography,and

computed tomographic scan

4. colonoscopy used to pickup the pathological specimen.

5. Polymerase chain reaction of ascitic fluid analysis was positive for Mycobacterium tuberculosis.

Ascitic fluid examination reveals the following features

, serum ascitis albumin gradient less than 1.1 g/dl,

straw coloured fluid with high protein

predominant cells are lymphocytic cells,

adenosine deaminase levels above 36 U/l is an diagnostic criteria for abdominal tuberculosis

Laparoscopy is also a useful investigation ..

Medical Management is with conventional antitubercular therapy for 6 months to 8 month duration

SURGICAL MANAGEMENT:

Definitive surgical procedure like resection and anastomosis, stricturoplasty and right hemicolectomy are surgical options.

A CASE OF ABDOMINAL TUBERCULOSIS WITH ILEAL TUBERCULOSIS WHICH SHOW ADHESIONS WITH GRANULOMATOUS LESION

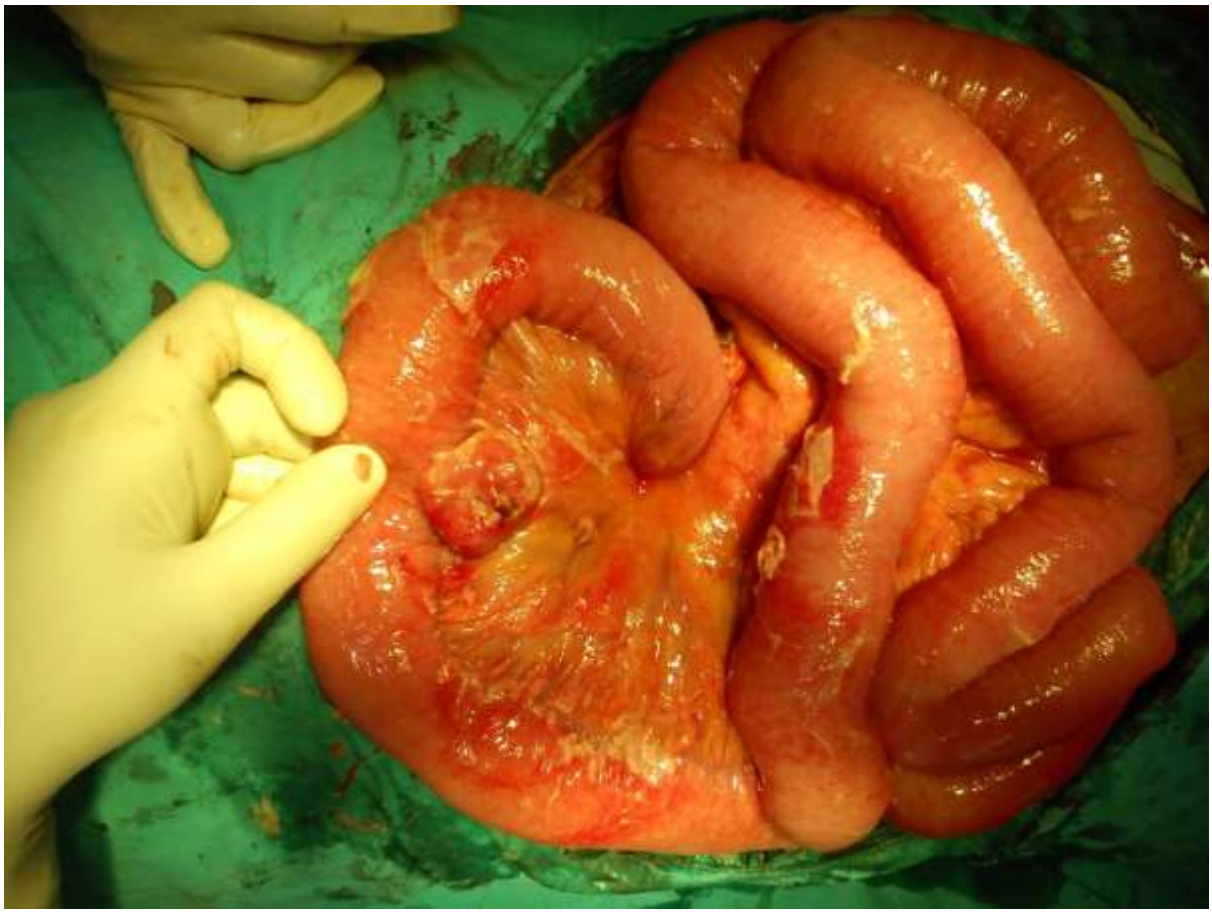


Fig 23: **a case of abdominal tuberculosis with ileal tuberculosis which show adhesions with granulomatous lesion**

CRITERIA FOR CHOOSING THE PATIENT TO BE INCLUDED IN THE STUDY

Inclusion criteria for this study:

a. clinically and radiological diagnosed

Hollow viscus perforation patient

b.age criteria between >15yrs and <60 yrs of age

c.patient taken for emergency laparotomy

d.patient observed in Sicu and post op ward

e.In post operative ward patient observed regarding

f. Patient willing for six month follow up.

g. After discharged, patient undergone readmission for same problem .

MATERIALS AND METHODS

Selectively, 60 patients are taken from Govt Kilpauk Medical college and Govt Royapettah Hospitals with effect from dec 2012 to july 2013

Patient are observed from casualty and in-patient department

Inclusion criteria for this study:

a. clinically and radiological diagnosed Hollow viscus perforation patient

b.age criteria between >15yrs and <60 yrs of age

c.patient taken for emergency laparotomy

d.patient observed in sicu and post op ward

e.In post operative ward patient observed regarding

1.Day of oral feed started

2. Is there any surgical site infection occurred

3. Patient undergone for wound gaping & secondary suturing

4. Patient fecal fistula occurred or not

5.Is there any mortality out come

D .Patient willing for six month follow up .After discharged patient undergone readmission for same problem .

OBSERVATIONS AND DATA ANALYSIS

VARIOUS INCIDENCE OF HOLLOW VISCUS PERFORATION

IN KMC & ROYAPETTEH G.H.

Duodenum	17
Ileum	18
Jejunum	10
Gastric	13
Colon	02
Total	60

DUODENAL INCIDENCE 28.33%

ILEAL INCIDENCE 30%

JEJUNUM INCIDENCE 16%

GASTRIC OUT COME 21.5%COLON INCIDENCE 3.3%

INCIDENCE OF VARIOUS HOLLOW VISCUS PERFORATION

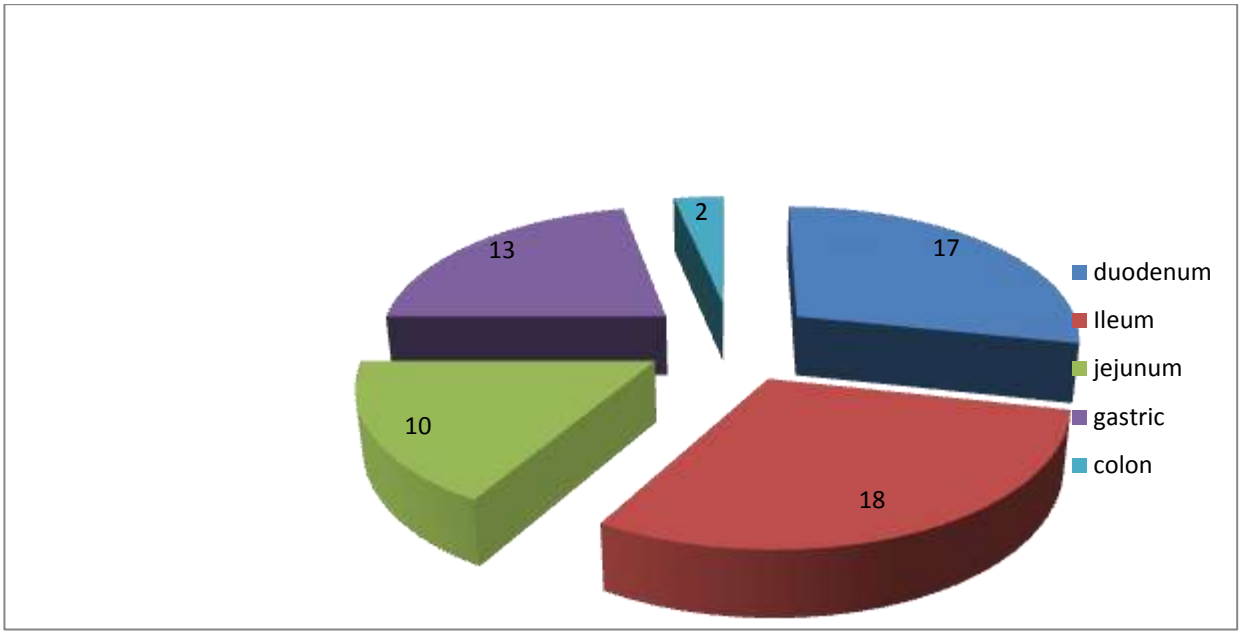


Figure 24: pie chart showing various incidence of hollow viscus perforation

Sex ratio of hollow viscus perforation

Male	46	76.6%
Female	14	23.4%

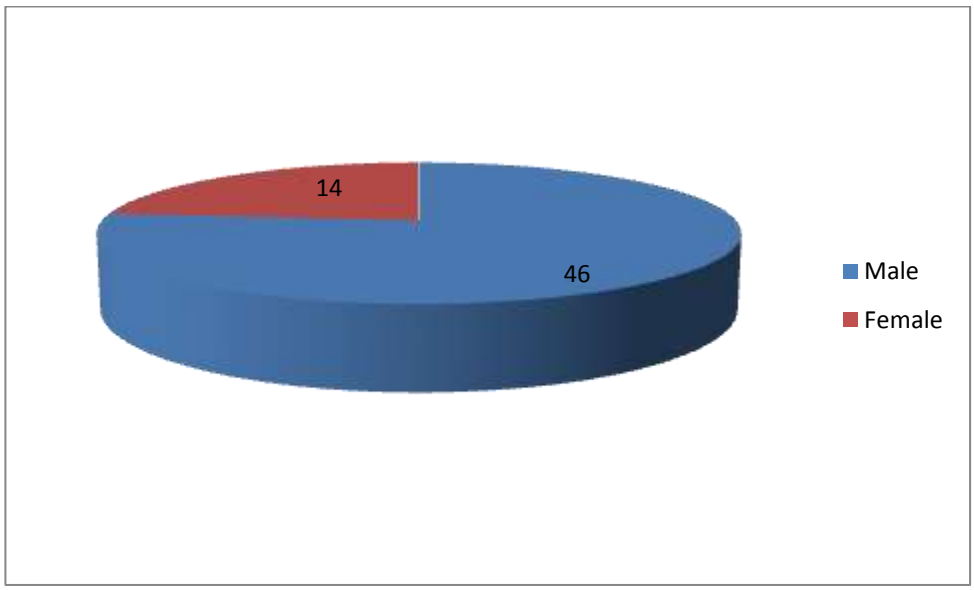


Fig no.25 sex ratio of hollow viscus perforation

MALE: FEMALE RATIO 3.5:1

Age wise incidence of hollow viscus perforation

15-20 yrs	**8**
21-30 yrs	**9**
31-40 yrs	**13**
41-50 yrs	**17**
➢ **50 yrs**	**13**

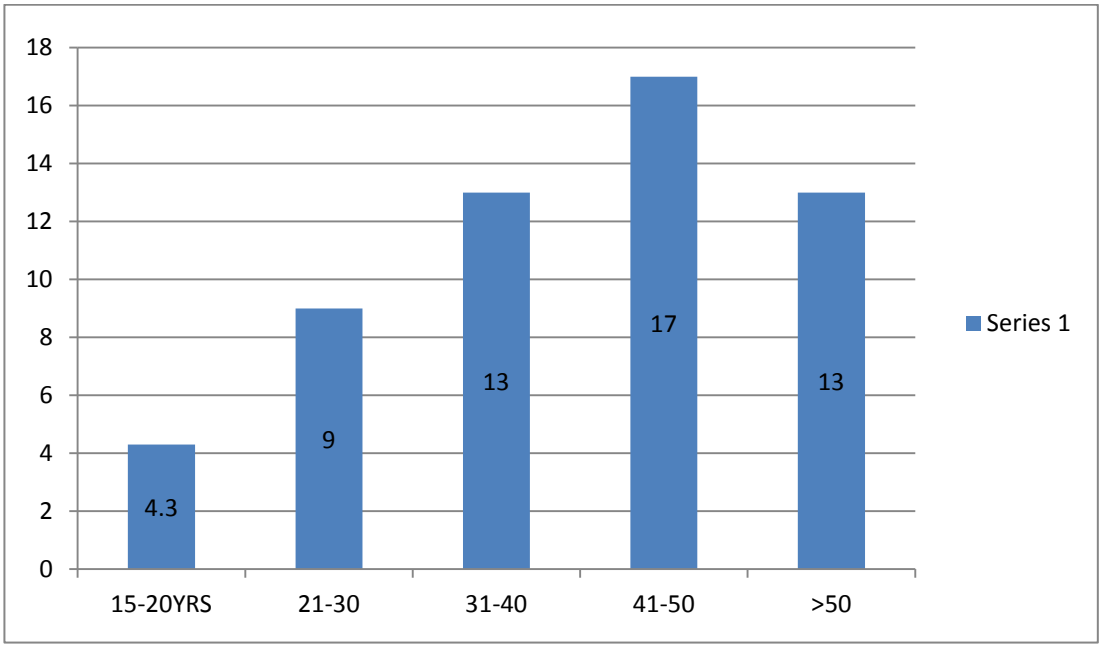


Fig no 26.showing the age wise incidence

Major morbidity in between 3rd and 4th decade

STATISTICS WISE ANALYSIS

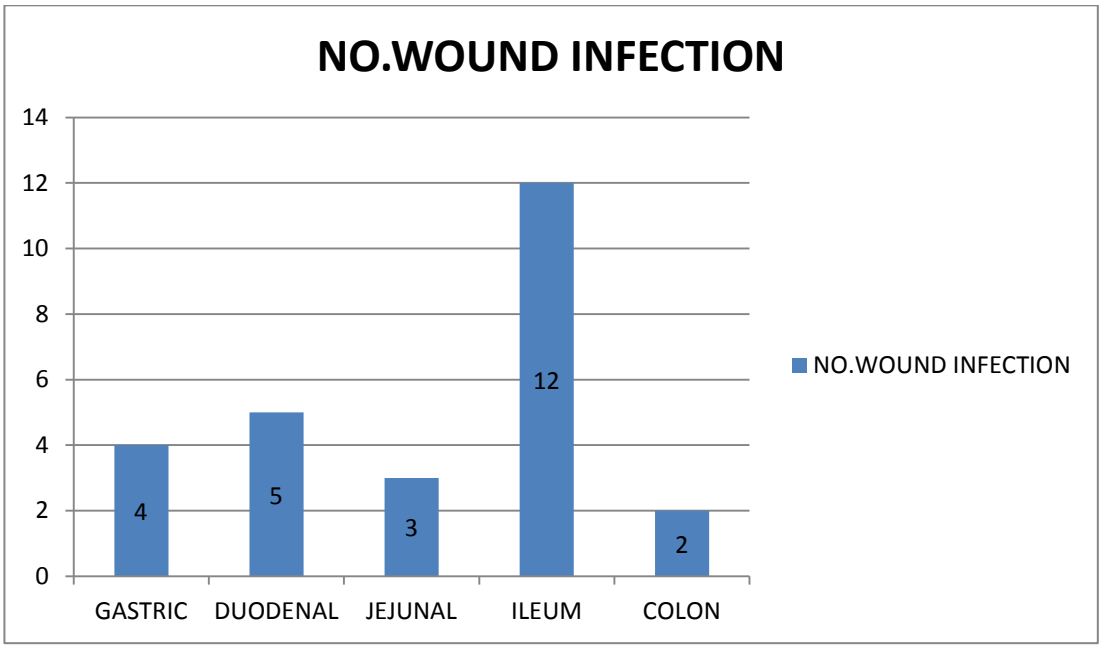


Fig no. 27

GASTRIC 4/13

DUODENUM5/17

JEJUNUM3/10

ILEUM12/18

COLON2/2

WOUND GAPING

Fig no 28 . WOUND GAPING

One of complication of ileal perforation

Pt is on 3rd week hospital stay

Single ileal perforation vs multiple ileal perforation

Single	**13**
Multiple	**5**

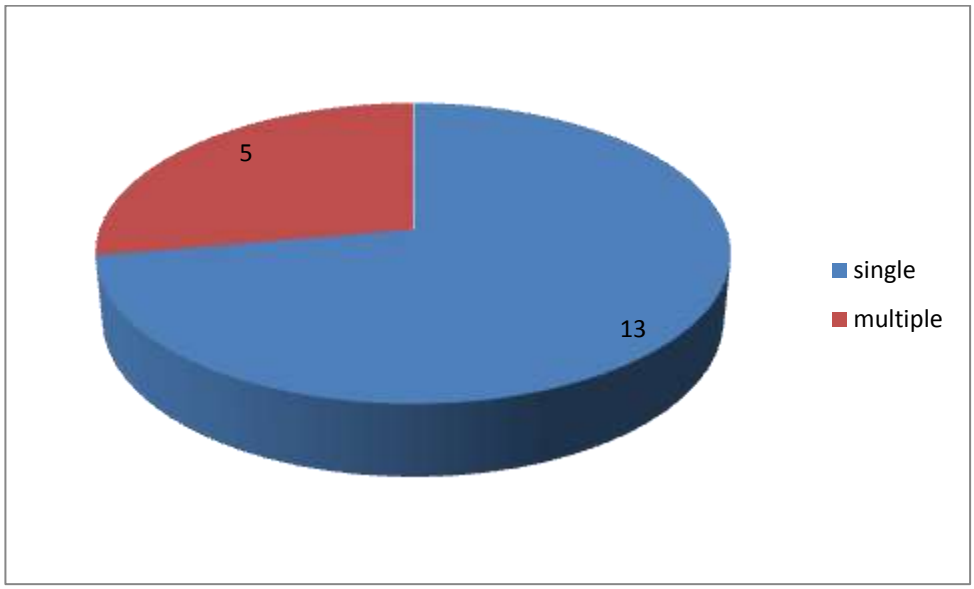
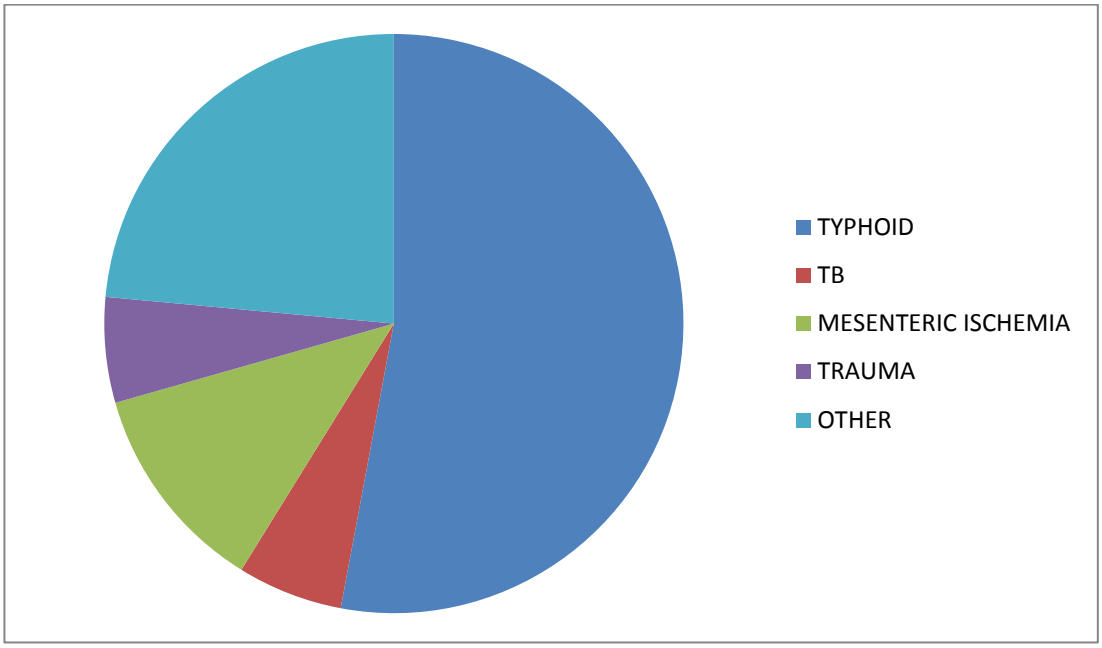


Fig no. 29.Incidence showing single & multiple ileal perforation

Etiological factors of ileal perforation

typhoid	**9**
Tuberculosis	**2**
Mesenteric ischaemia	**2**
trauma	**1**
others	**4**



NO. OF PATIENT UNDERGOING SECONDARY SUTURING

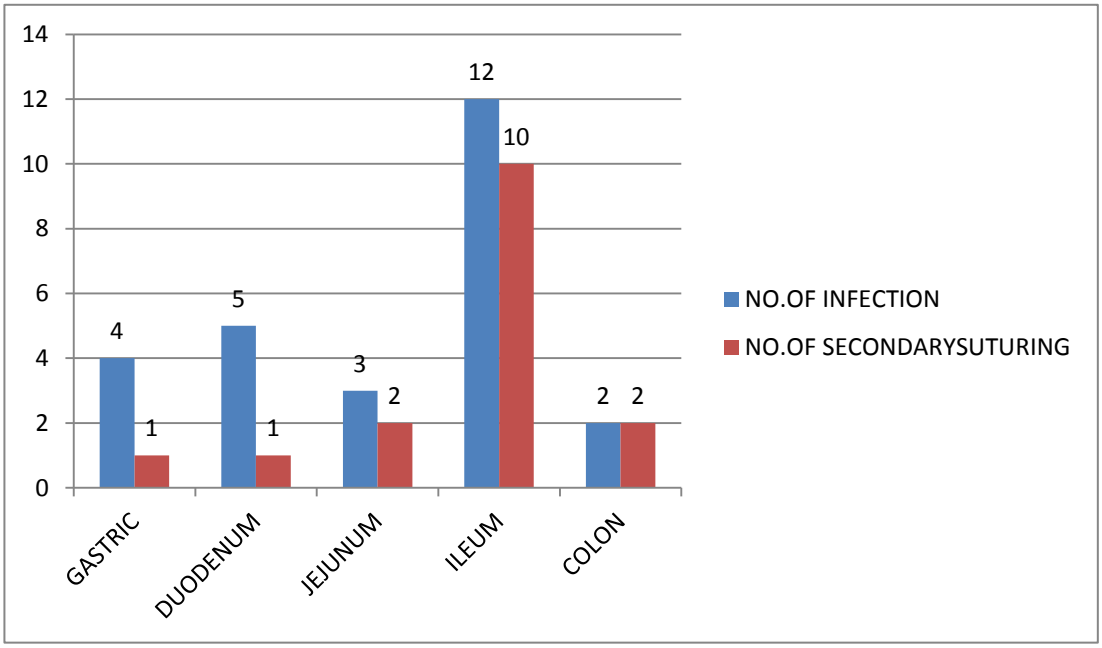


Fig no. 30

MORTALITY RATE AMONG HOLLOW VISCUS PERFORATION

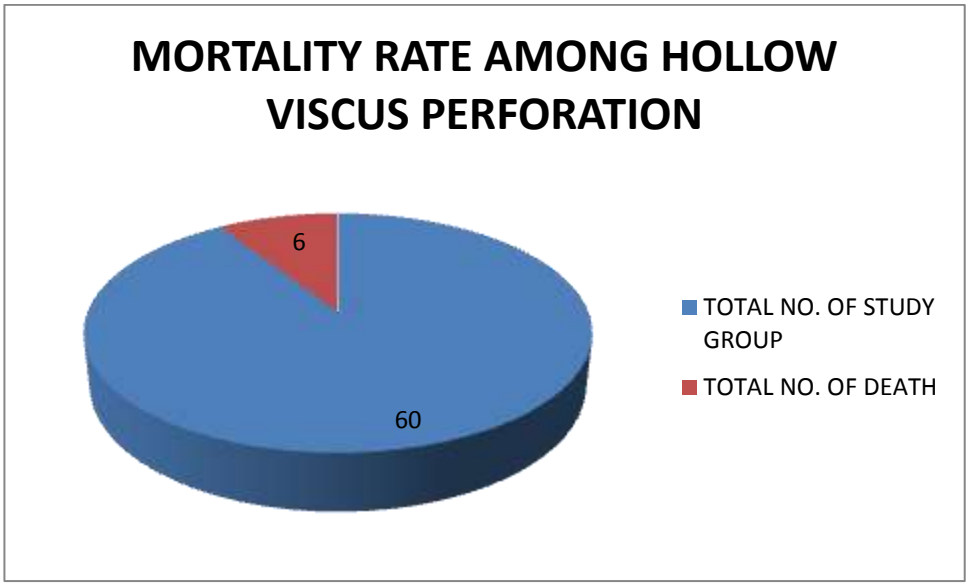


Fig no .31

INCIDENCE OF DEATH AMONG

HOLLOW VISCUS PERFORATION

Ileum	**4**
Duodenum	**2**

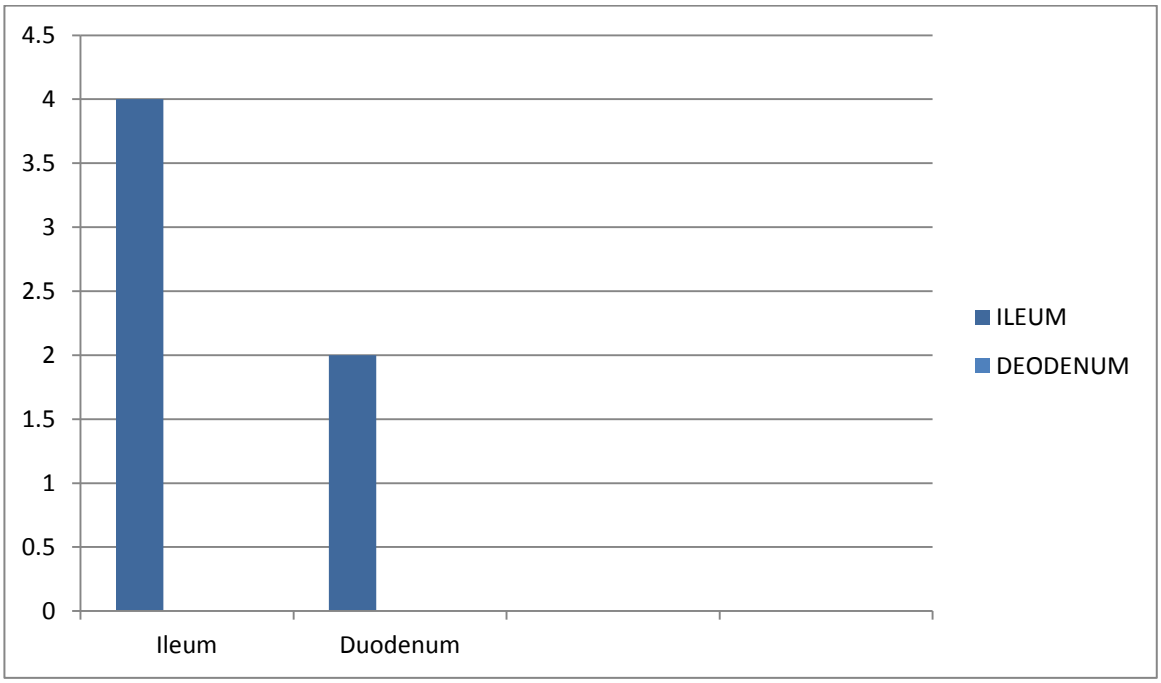


Fig no.31

PERFORATION VS SMOKING OUTCOME

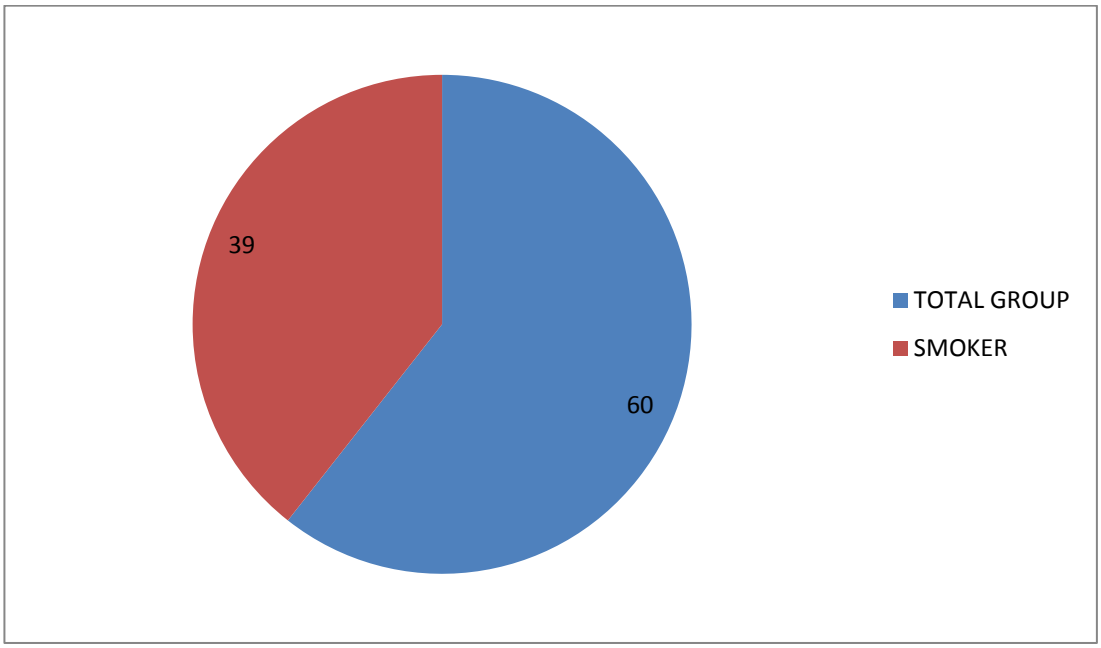
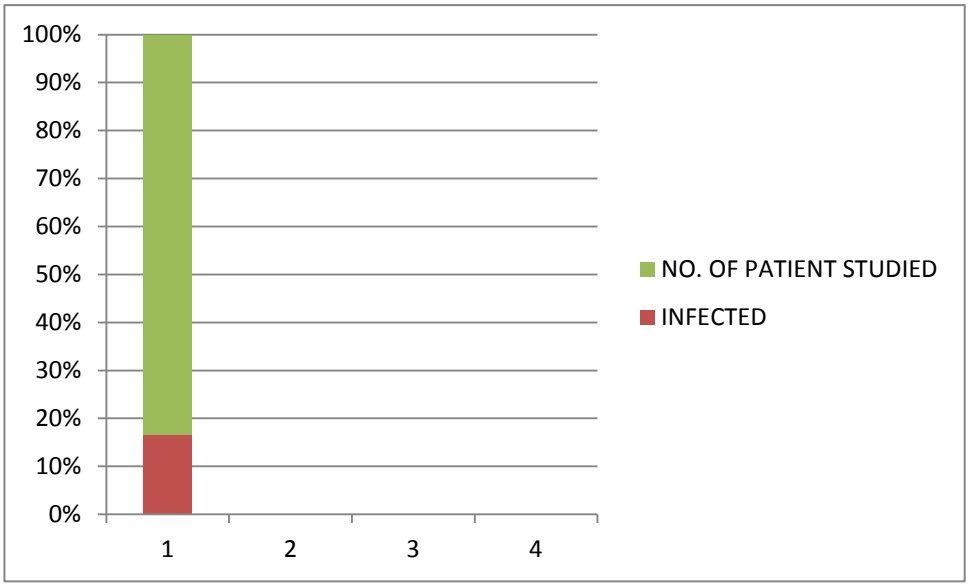


Fig no. 32

NO. OF PATIENTS WITH WOUND INFECTION

Total no. of patient studied **60**

Total no. of patient undergone wound infection **26**



VARIOUS DATA OF WOUND INFECTION

Types of perforation	Total no. of patient	No. of patient woynd infected
Gastric	13	4
Duodenum	17	5
Jejunum	10	3
Ileum	18	12
Colon	2	2

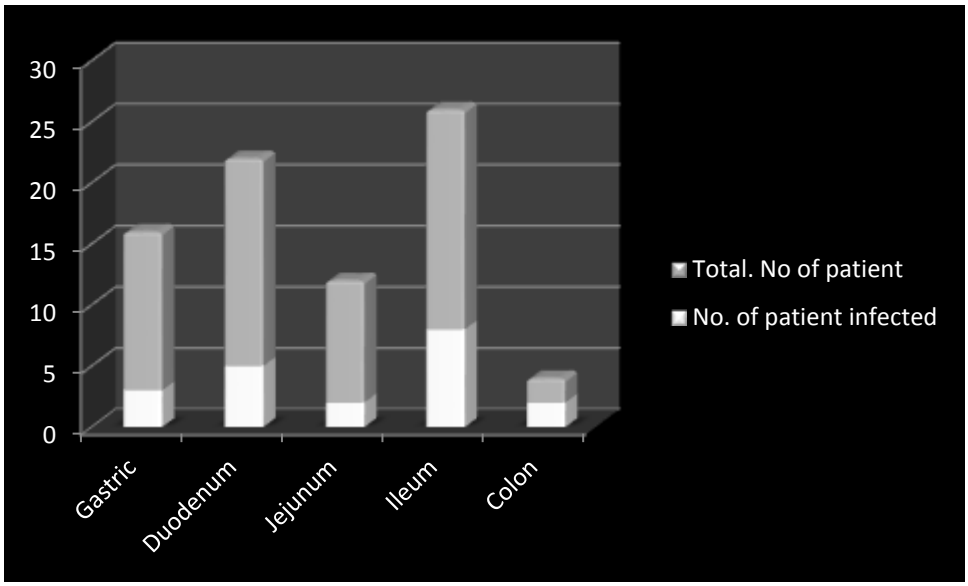


Fig no. 33

TOTAL NO. OF PATIENT WHO UNDERWENT SECONDARY SUTURING

Total no. of patient studied	**60**
No. of patient wound become infected	**25**
No. of patient undergone secondary suturing	**17**

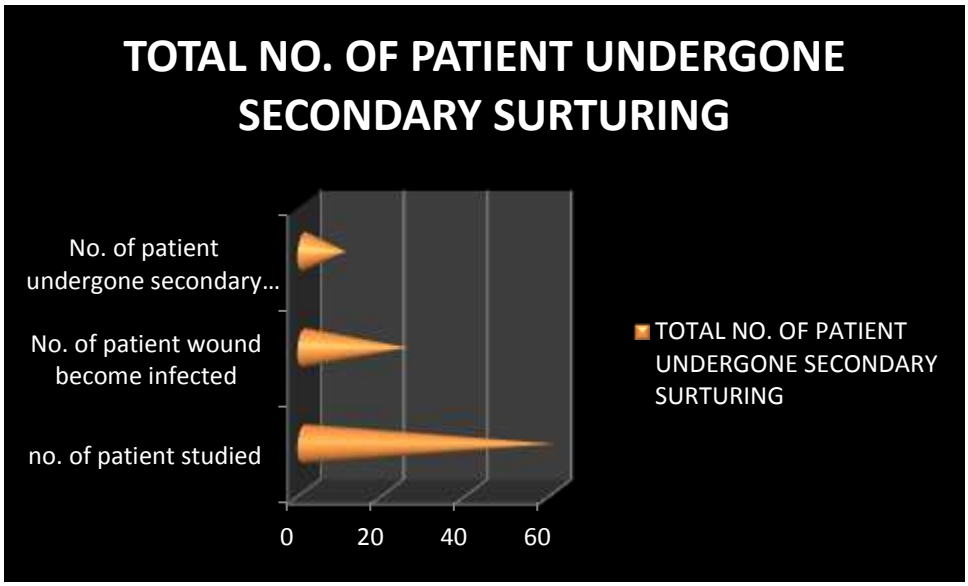


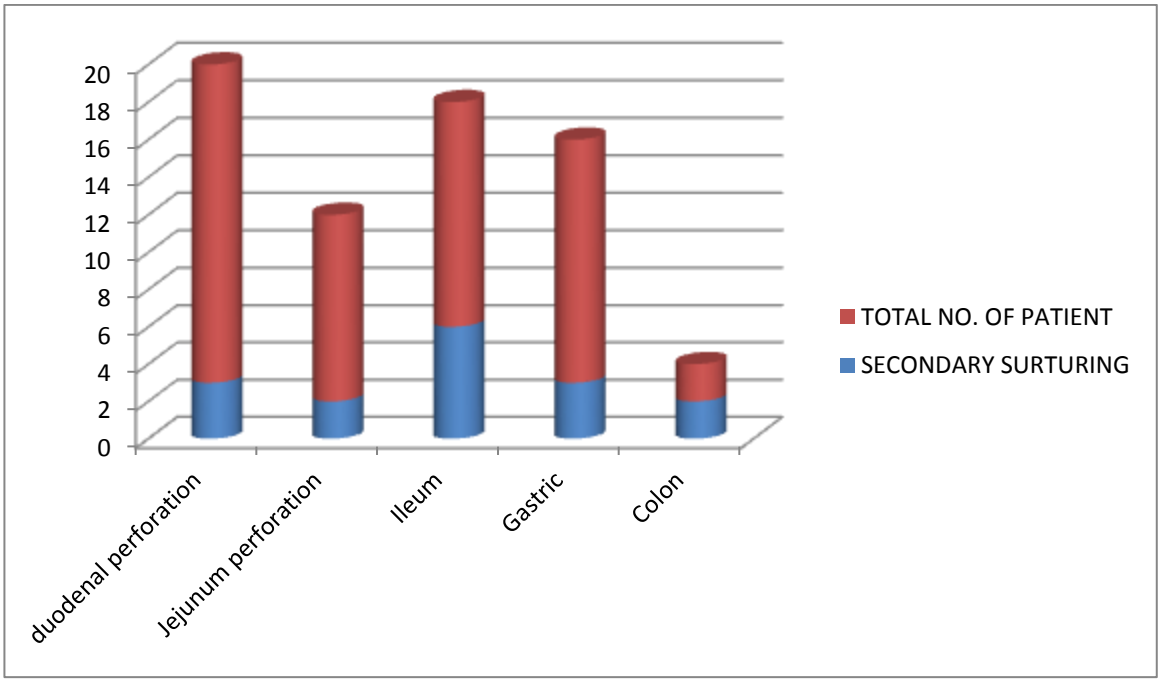
Fig no.34

Summary of previously reported series of small intestinal perforation [8 - 17]

Author [Ref]	Total Cases	Typhoid Perforations (%)	Non-specific Ulcer Perforation (%)	Tubercular Perforations n (%)	Morta-lity
Khan 2004	18	7 (38.9)	5 (27.8)	2 (11.1)	NA
Chaterjee 2001, 2003	460	248 (53.9)	111 (24.1)	16 (3.5)	20.9%
Chitkara 2002	216	92(42.6)	36(16.7)	36(16.7)	11.5%
Ray 2001	30	8(26.7)	5(16.7)	4(13.3)	6.7%
Chulakamoni 1996	8	2 (25)	1 (12.5)	0	0
Dorairajan 1995	103	69 (66.9)	7 (6.8)	13 (12.6)	NA
Sharma 1991	62	42 (67.7)	5 (8.1)	12 (19.3)	11.3%
Bose 1986	75	46 (61.33)	1 (1.3)	8 (10.6)	16%
Khanna 1984	125	100 (80)	0	4 (3.2)	NA
Nadkarni 1981	32	8(25)	18(56.2)	3(9.3)	28.1
Mehendale 1979	32	9(28.1)	2(6.2)	13(40.6)	37.5%
MY STUDY	**18**	**9**	**7**	**2**	**4 (22) %**

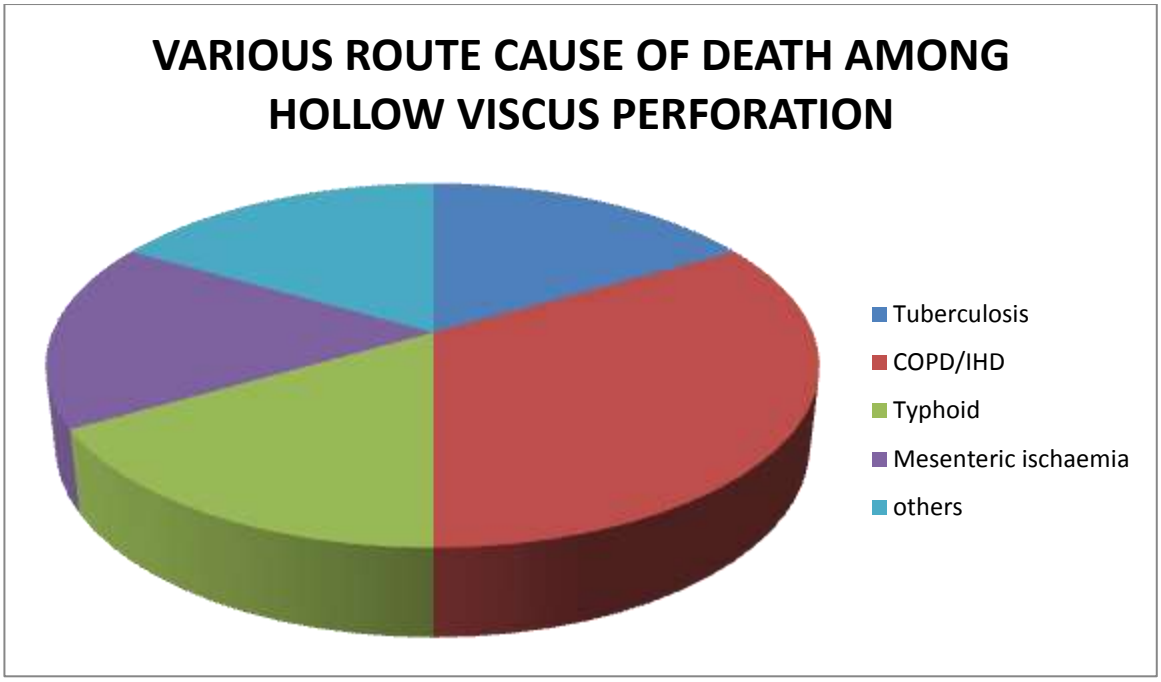
DATA OF SECONDARY SURTURING

SITE OF PERFORATION	TOTAL NO.	SECONDARYSURTURING
Duodenal perforation	17	3
Jejunum perforation	10	2
Ileum	18	12
Gastric	13	3
Colon	2	2



VARIOUS ROUTE CAUSE OF DEATH

Etiology	Route cause	No. of death
Tuberculosis	Septicemia	1
COPD/IHD	Cardio respiratory failure	2
Typhoid	Septicemia shock	1
Mesenteric ischemia	Septicemia shock	1
Others	Septicemia shock	1



CONCLUSION

According to the data collected from Kmch and Royapettah hospital .Totally 18/60 is ileal perforation which is 30% of total participation and first among the hollow viscus perforation. The colonic perforation case report was minimal which only participate only 3.5% of total incidence.

Most commonly 3rd and 4th decade's people are more vulnerable for perforation. Smoking, alcohol, stress, and NSAIDs, gram negative salmonella were take participation in hollow viscus perforation.

Other not uncommon factors are ischemic bowel disease, trauma, lymphoma are also take participation in hollow viscus perforation.

In 7 patient of the 34 who were smoker in this study ileal patient as noted.

Totally 25 cases wound became infected out of 60 cases.

The wound infected patient have to be stay in hospital for more days.

The DALY also more. The most common organism responsible for wound infection is Staphylococcus aureus second most common organism was klebsiella.

Among 25 infected patients 13 patients are associated with ileal perforation.among those 10 had to secondary suturing.

Even though trauma also be an important factor for ileal perforation trauma associated perforation cause haemoperitoneum but does not common cause of peritonitis due earlier intervention and does cause minimal wound infection.

In my study any patient won't undergo for entero - cutaneous fistula. Totally 60 patient only 4 patient had died of which total 3 patient were due to septic shock, remaining patient due to cardio respiratory failure.

Among ileal perforation typhoid is an important factor for ileal perforation. Which will commonly happened in 3-4th week of infection. In our territory, poor water contamination , unhygienic food plays an important role in typhoid etiology . The most common organism were s.typhi, s.paratyphiA and b are major organism for typhoid associated ileal perforation.

TUBERCULOSIS is an important factor for ilel perforation.in our Indian country abdominal tuberculosis is an most common association for multiple ileal perforation. DM / HIV, low socio economic status , are major triggering factor for tuberculosis spreading status. Among

hollow viscus perforation ileal perforation is the major cause of more wound infection.

In our institute study, totally 6/60 patient land up with death. Among six death four patient due to ileal perforation. Other two patient death due to duodenal perforation. The major root cause of death was septcemia induced shock.

ABSTRACT

TITLE:incidence and outcome of ileal perforation among hollow viscus perforation

KEY WORDS: incidence, ileum, viscus., perforation, typhoid, duodenum,jejunum .

Introduction : World wide hollow viscus perforation was common emergency problem in surgical casualty department . incidence of hollow viscus perfartion is variable from regional wise statistics. But all over worldwide Male incidence was greater than female incidence.

male:female ratio was 4:1.

In my study sex ratio out come is 3.5:1. Life style modification , stress free life regularized food habit all will be a deciding factor for hollow viscus perforation

DISCUSSION: A study was conducted in Govt Kilpauk medical College Hospital **and** Govt royapettah hospital by assessing the 60 cases of hollow viscus perforation .Among sixty patient 14 patient was female and 46 patient was male.Patient are included in this study based on clinical and radiological based evidence.Patient taken for emergency laparotomy procedure , closing of perforation by Primary closure ,

resection and anastomosis, patch closure.postoperatively patient kept in Sicu and surgical post operative ward .

Post operative followup based on regarding day of oral feeds started , surgical wound infection, wound gaping and those patient undergone for secondary suturing.Among ileal perforation 8/18 patient wound became infected .5/8 infected ileal perforation wound undergone secondary suturing.

Those patient undergone for secondary suturing their DALY (daily adjusted life year) was more. Data analysed regarding food habits of every patient .

Those patient had an hotel food habits , smoking, alcoholism, stress ful life they became as victim for hollow viscus perforation.

The common age groups are 3^{rd} and 4^{th} decades. Among sixty patient 24 patient belongs to 3^{rd} and 4^{th} decades. Ileal perforation due to typhoid is an important etiological factor at 3^{rd} and fourth week of infection .Widal test and HPE is an important test to rule out other etiological factors of perforation.

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Name	Age	Sex	Abd. Pain	Abd. Dist.	Fever	Vomi-ting	Bowel habits	Smoking H/O	NSAID intake	Alcohol H/O	Co-Morbid	Food Habits	X-ray	USG	Site of perforation	Other finding	Special Investigations	Procedure Done	Wound Infection	Gaping/ Secondar	DT removed on	Orals started on	II Surg	Disch. on	Follow-up
PALANI	44	M	Y	N	Y	Y	D	Y	Y	Y	NIL	HOME	G+	NT	ILEUM**	NIL	HPE-TB	L	Y	Y	4TH POD	4TH POD	NIL	12TH P	NIL
VENKETESH	32	M	Y	N	N	Y	N	Y	Y	Y	NIL	HOTEL	G+	FF+	GASTRIC	NIL	HPE	L	N	N	4TH POD	4TH POD	NIL	10THPO	NIL
MUNIAMMAL	50	F	Y	Y	N	Y	N	N	Y	N	DM	HOME	G+	NT	DUODENAM	DIVERTICULUM	NIL	L	Y	N	6TH POD	5TH	NIL	15TH P	RE-ADM.
VIJEYANTHY	35	F	Y	Y	Y	Y	D	N	N	N	NIL	HOME	G+	FF+	JEJUNAM	NIL	NIL	L	N	N	5THPOD	5THPOD	NIL	10THPO	NIL
DEVARAJAN	62	M	Y	Y	N	Y	N	Y	N	Y	HT	HOTEL	G+	FF+	DUODENAM	NIL	NIL	L	N	N	4TH POD	4TH POD	NIL	10THPO	NIL
SENTHIL KUMA	32	M	Y	Y	Y	Y	D	N	Y	N	NIL	HOTEL	G+	FF+	ILEUM	NIL	WIDAL POSITI	L	Y	Y	4TH POD	4TH POD	NIL	7THPOI	NIL
RAJESH	18	M	Y	N	N	Y	N	Y	Y	Y	NIL	HOTEL	G+	FF+	GASTRIC	NIL	HPE	L	N	N	3RD POD	3RD POD	NIL	8TH PO	READM.
GOPAL	60	M	Y	Y	N	Y	N	Y	Y	Y	DM/HT	HOME	G+	NT	DUODENAM	NIL	NIL	L	Y	N	4TH POD	4TH POD	NIL	10POD	NIL
SIVA	48	M	Y	N	N	Y	N	Y	N	Y	HT	HOME	G+	FF+	DUODENAM	NIL	NIL	L	Y	N	4TH POD	4TH POD	NIL	10TH P	NIL
VARADHAN	55	M	Y	Y	Y	Y	C	Y	N	Y	DM	HOME	DILA	FF+, DILA	ILEUM	MS*	PT/INR	L	Y	Y	NR		YES	DEATH	13POD
PARISIDHU	27	M	Y	Y	Y	Y	D	Y	N	Y	NIL	HOTEL	G+	FF+	ILEUM	NIL	WIDAL POSITI	L	Y	Y	5THPOD	5THPOD	NIL	12TH P	NIL
SURESH	22	M	Y	N	N	Y	N	Y	Y	Y	NIL	HOTEL	G+	FF	GASTRIC	NIL	HPE	L	N	N	4TH POD	4TH POD	NIL	8TH PO	NIL
VENKETESH	40	M	Y	Y	N	Y	D	Y	Y	Y	HT	HOTEL	G+	FF+	DUODENAM	NIL	NIL	L	N	N	4TH POD	4TH POD	NIL	10THPO	NIL
JEYARAJ	42	M	Y	Y	N	Y	N	Y	Y	Y	NIL	HOME	G+	FF+	JEJUNAM	NIL	NIL	L	Y	Y	6TH POD	6THPOD	NIL	11THPO	READM.
RAVI	46	M	Y	N	N	Y	D	Y	Y	N	NIL	HOTEL	G+	NT	GASTRIC	NIL	HPE	L	Y	N	4TH POD	6THPOD	NIL	7THPOI	NIL
BABU	56	M	Y	N	Y	Y	N	Y	Y	Y	HT	HOTEL	G+	FF+	GASTRIC	NIL	HPE	L	N	N	5TH POD	5THPOD	NIL	8TH PO	NIL
BABU	27	M	Y	N	N	N	N	N	N	Y	NIL	HOME	G+	BLOODY	*JEJUNAM*	TRAUMA	NIL	L	N	N	4TH POD	4TH POD	NIL	20THPO	NIL
GUNASEKARAN	44	M	Y	Y	Y	Y	D	Y	Y	Y	NIL	HOME	G+	FF+	JEJUNAM	NIL	NIL	L	N	N	4TH POD	4TH POD	NIL	10TH P	NIL
SRINIVASAN	54	M	Y	Y	N	Y	N	N	N	N	NIL	HOME	G+	FF+	DUODENAM	NIL	NIL	L	Y	Y	4TH POD	4TH POD	NIL	15TH P	READM.
FARIDHA	48	F	Y	Y	Y	Y	N	Y	N	Y	T/DM/IH	HOME	G+	FF+	DUODENAM	NIL	NIL	L	N	N	NOT AVAILABLE		NIL	1ST POD	DEATH
SATHYAPRIYA	17	F	Y	Y	Y	Y	D	N	Y	N	NIL	HOME	G+	FF+	ILEUM**	>1PERFORATION	HPE-TB	L	Y	Y	7TH POD	7THPOD	NIL	18 POD	READM.
MADHIPRASATH	30	M	Y	N	Y	N	N	N	N	Y	NIL	HOTEL	N	FF+	DUODENUM	NIL	NIL	L	N	N	4TH POD	4TH POD	NIL	10thPOI	NIL
MARIAMMAL	46	F	Y	Y	Y	Y	N	Y	N	Y	NIL	HOTEL	G+	FF+	GASTRIC	NIL	HPE	L	N	N	4TH POD	4TH POD	NIL	12TH P	NIL
REVATHY	17	F	Y	Y	Y	Y	C	N	N	N	TB	HOME	N	DILATED	ILEAL	GANGREN S	NIL	L	N	N				DEATH	3POD
PARTHASARATH	48	M	Y	Y	Y	Y	D	Y	Y	N	NIL	HOME	G+	FF+	ILEAL	NIL	WIDAL+	L	Y	Y	5THPOD	5THPOD	NIL	13THPO	NIL
USHA	35	F	Y	Y	Y	Y	N	N	N	N	DM	HOME	G+	FF+	ILEAL	TRAUMA	WIDAL-	L	Y	N	4TH POD	4TH POD	NIL	10THPO	NIL
KARUNAKARAN	63	F	Y	Y	Y	N	N	N	N	N	COPD	HOME	G+	FF+	DUODENAL	NIL	NIL	L	N	N	PT DEATH	AT 4TH	POD		
KUMAR	40	M	Y	N	Y	Y	N	Y	Y	Y	NIL	HOTEL	G+	FF+	GASTRIC	NIL	HPE	L	N	N	4THPOD	4TH POD	NIL	12TH P	NIL
PAKASMANDAL	35	M	Y	N	Y	Y	N	Y	Y	N	NIL	HOTEL	G+	NT	GASTRIC	NIL	HPE	L	N	N	4THPOD	4THPOD	NIL	8TH PO	NIL
RAJKANNAN	43	M	Y	Y	N	Y	D	Y	N	Y	NIL	HOTEL	G+	NT	DUODENAM	NIL	NIL	L	N	N	4THPOD	4THPOD	NIL	9THPOI	NIL
ABIRAMI	18	F	Y	Y	Y	Y	Y	N	N	N	NIL	HOME	G-	FF+	DUODENUM	NIL	NIL	L	N	N	5THPOD	5THPOD	NIL	9THPOI	NIL
KALA	19	F	Y	Y	Y	Y	D	N	N	N	NIL	HOME	G+	FF+	JEJUNAM	NIL	NIL	L	N	N	4THPOD	4THPOD	NIL	9THPOI	NIL
RAJA	46	M	Y	N	Y	N	D	Y	Y	N	HD	HOTEL	G-	FF+	COLON*	NIL	NIL	L	Y	Y	7THPOD	7THPOD	NIL	20THPO	NIL
SANTHOSH	25	M	Y	Y	Y	Y	C	Y	Y	Y	NIL	HOTEL	G-	FF+	JEJUNAM	NIL	NIL	L	Y	Y	5THPOD	5THPOD	YES	13THPO	NIL
RAJESH	25	M	Y	Y	Y	Y	N	Y	Y	Y	NIL	HOTEL	G+	FF+	DUODENUM	NIL	NIL	L	N	N	4THPOD	4THPOD	NIL	9THPOI	NIL
GANESH	36	M	Y	N	Y	Y	N	Y	N	N	NIL	HOTEL	G+	FF+	GASTRIC	NIL	HPE	L	N	N	5THPOD	5THPOD	NIL	11THPO	NIL
RAMESH	34	M	Y	Y	Y	Y	N	Y	Y	N	NIL	HOTEL	G-	FF+	JEJUNAM	DIVERTICULAM	NIL	L	N	N	4THPOD	4THPOD	NIL	9THPOI	NIL
GANGAN	43	M	Y	Y	Y	Y	D	N	N	Y	DM	HOTEL	G-	FF+	ILEUM	NIL	WIDAL+	L	Y	Y	5THPOD	5THPOD	NIL	14THPO	NIL
MADHU	48	M	Y	Y	Y	N	N	Y	Y	Y	HD	HOME	G+	FF+	ILEUM	NIL	WIDAL-	L	Y	Y	6THPOD	6THPOD	NIL	12THPO	NIL

DEVARAJAN	36	M	Y	Y	Y	N	N	N	Y	Y	NIL	HOTEL	G+	FF+	DUODENUM	NIL	NIL	L	N	N	4THPOD	4THPOD	NIL	10THPO	NIL
KOLAN	55	M	Y	Y	Y	N	N	Y	N	Y	DM	HOME	G-	FF+	COLON	NIL	NIL	L	Y	Y	7THPOD	9THPOD	NIL	22THPO	Nil
RAJ	55	M	Y	Y	Y	Y	D	Y	Y	N	DM	HOME	G+	FF+	JEJUNAM	NIL	NIL	L	Y	Y	5THPOD	5THPOD	NIL	9THPOI	NIL
SIVAM	37	M	Y	Y	Y	N	N	Y	Y	Y	HD	HOME	G+	FF+	ILEUM	NIL	WIDAL NEGAT	L	Y	Y	6THPOD	6THPOD	NIL	15THPO	NIL
SEZLIAN	49	M	Y	Y	N	Y	N	Y	N	Y	NIL	HOTEL	G+	FF+	GASTRIC	NIL	NIL	L	Y	N	4THPOD	4THPOD	NIL	9THPOI	NIL
KUPPAN	55	M	Y	Y	Y	N	N	Y	N	Y	NIL	HOME	G+	FF+	GASTRIC	NIL	HPE	L	N	N	4THPOD	4THPOD	NIL	12THPO	NIL
SUSEELA	39	F	Y	Y	Y	N	D	Y	Y	N	NIL	HOME	G-	FF+	ILEUM	NIL	WIDAL+	L	N	N	4THPOD	4THPOD	NIL	10THPO	NIL
SANJAY	58	M	Y	Y	N	N	N	Y	Y	N	NIL	HOME	G+	FF+	GASTRIC	NIL	NIL	L	Y	N	3THPOD	4THPOD	NIL	9THPOI	NIL
KANNAN	48	M	Y	Y	Y	Y	N	Y	Y	N	NIL	HOTEL	G_	FF+	DUODENUM	NIL	NIL	L	N	N	3RD POD	3RD POD	NIL	10THPO	NIL
RAMATHAL	40	F	Y	Y	Y	N	C	Y	Y	Y	NIL	HOTEL	G-	FF+	ILEUM	NIL	WIDAL+	L	Y	Y	6THPOD	6THPOD	NIL	20THPO	Nil
UMESH	55	M	Y	Y	Y	Y	D	N	N	N	NIL	HOME	G-	FF	ILEUM	NIL	WIAL	L	Y	Y	5THPOD	5THPOD	YES	23POD	NIL
KAMESH	52	M	Y	N	Y	N	N	Y	N	Y	NIL	HOTEL	G+	FF+	DUODENUM	PW	NIL	L	N	N	4THPOD	4THPOD	NIL	10THPO	NIL
MANI	38	M	Y	Y	N	N	D	Y	N	Y	NIL	HOME	G+	FF/DILAT	ILEUM	NIL	WID	L	N	N	4THPOD	4THPOD	NIL	9THPOI	NIL
DHANABAL	55	M	Y	Y	Y	Y	C	N	EA	Y	NIL	HOME	G+	FF+	ILEUM	NIL	WIDAL	L	Y	Y	7THPOD	7POD	NIL	25TH	READM.
RAJESH	55	M	Y	Y	Y	Y	D	Y	Y	Y	NIL	HOTEL	G+	FF+	DUODENUM	NIL	WIDAL+	L	Y	N	4THPOD	4THPOD	NIL	12THPO	NIL
KANIMOZHI	30	F	Y	Y	Y	Y	D	N	N	N	DM	HOME	G-	FF+	ILEUM	NI	WIDAL +	L	N	N	5THPOD	5THPOD	NIL	12THPO	NIL
DHAYALU	50	F	Y	N	Y	N	N	N	Y	N	HT	HOME	G+	FF+	GASTRIC	NIL	HPE	L	N	N	4THPOD	4THPOD	NIL	09THPO	NIL
MURALI	28	M	Y	Y	Y	Y	N	Y	Y	N	NIL	HOTEL	G+	FF+	DODENUM	NIL	NI;L	L	N	N	4THPOD	4THPOD	NIL	9THPOI	NIL
SULOCHANA	48	F	Y	Y	Y	Y	D	N	N	N	NIL	HOME	G+	FF+	JEJUNUM	NIL	NIL	L	N	N	5THPOD	5THPOD	NIL	12POD	NIL
KIRANI	19	F	Y	Y	Y	Y	D	N	N	N	NIL	HOME	G+	FF+	ILEUM	NIL	WIDAL +	L	N	N	4THPOD	4THPOD	NIL	10THPO	NIL
								DM--DIABETES TOTAL NO OF STUDY GROUP---60								ILEUM--18	MS* MESENTERIC ISCHEMIA								D
								HT -HYPERTENSIONTOTAL NO OF MALE					MAL	34		GASTRIC--13	ILEUM** ILEUM TB								
								G-GAS UNDER DIAPRACTOTAL NO. OF FEMALE PA						16		DUODENUM--17									
								FF- FREE FLUID								JEJUNUM 8									
								USG -ULTRASOUND																	
																POSTERIORWALL	PW								
									Y-YES																
									N-NO																